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The Role of Auditory Feedback for Speech Intensity Regulation in Parkinson's Disease

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Abstract

Hypophonia (low speech intensity) has been found to be the most common speech symptom experienced by individuals with Parkinson's disease (PD). Previous research suggests that, in the PD population, there may be abnormal integration of sensory information for motor production of speech intensity. In the current study, auditory feedback was systematically manipulated during sensorimotor conditions that are known to modulate speech intensity in everyday contexts. Twenty-six individuals with PD and twenty-four neurologically healthy controls were asked to complete the following tasks: converse with the experimenter with varying distances between the participant and listener (near and far distances), vowel prolongation, read sentences at a comfortable loudness, complete a magnitude production task (reading 2 times louder, 4 times louder, maximum loudness), and complete an imitation task (50dB, 60dB, 70dB, 80dB), while hearing their own speech intensity randomly altered. Altered intensity feedback conditions included 5, 10 and 15dB reductions and increases in the feedback intensity. Participants were also asked to read sentences with and without an instruction to attempt to ignore the auditory feedback. Speech tasks were completed in no noise, background noise, and a complete masking noise condition. Outcome measures included speech intensity (dB) and loudness perception ratings obtained using a visual analogue scale. Overall results indicate that individuals with PD display a reduced response to the altered intensity feedback in all speech tasks, suggestive of abnormal of processing of auditory feedback for speech intensity regulation. Specific deficits related to the perception of self-loudness are suggested based on the current findings. Clinical implications are discussed as they relate to understanding specific deficits of auditory processing for speech impairments in PD.

Keywords: Parkinson's disease, movement disorders, sensorimotor integration, auditory feedback, altered auditory feedback, auditory masking noise, speech intensity, loudness perception

Summary for Lay Audience

Approximately 80% of individuals with Parkinson's disease (PD) experience low speech intensity. Previous researchers have shown this speech problem has a negative impact on overall quality of life. The cause of this speech problem is unclear and this prevents appropriate therapy development. Producing speech intensity that is appropriate when communicating with others is a complex process, which involves regulating self-produced speech intensity, monitoring ambient or background noise in the surroundings, and maintaining speech loudness throughout a conversation. It is possible that the low speech intensity produced by individuals with PD is caused by a problem related to how they perceive the loudness of their own speech. To examine this potential cause, the current study systematically manipulated how individuals hear their own speech by altering auditory feedback. Testing involved making an individual's speech sound louder than was actually being produced and sometimes quieter than was actually being produced. Previous research suggests that healthy speakers compensate for this type of manipulation by producing speech in the opposite direction. For example, when an individual's speech is manipulated to sound louder than is actually being produced, the speaker typically adjusts their speech to be quieter. This testing was conducted while individuals were being asked to complete a variety of speech tasks typically encountered in daily life, such as in conversation and while speaking in background noise. Results from twenty-six individuals with PD and twenty-four neurologically healthy participants found that individuals with PD made significantly smaller adjustments in their speech intensity during altered auditory feedback conditions compared to the non-neurologically impaired participants. These findings suggest that in PD, there may be abnormal perception of the sound of their own speech and this abnormality may be related to the cause of their low speech intensity. Findings from this study are anticipated to impact how clinicians treat the speech problems in PD and may lead to the development of new therapeutic techniques.

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List of Abbreviations

AIF	Altered intensity feedback
BGN	Background noise
ConvN	Conversation at a near distance
ConvF	Conversation at a far distance
dB	Decibels
HC	Healthy controls
MP	Magnitude production
PD	Parkinson’s disease
Read	Reading task
SD	Standard deviation
VAS	Visual analogue scale

Chapter 1: Introduction

Parkinson's disease (PD) is a neurodegenerative movement disorder (Duffy, 2013; Hobson, 2003). The percentage of individuals over the age of 50 years with PD is 4% or an estimated 4.1-4.6 million worldwide in 2005, and projection analysis yielding an estimated 8.7-9.3 million by the year 2030 (Dorsey et al., 2007; Goetz & Pal, 2014). In Canada, prevalence of PD is 0.2% of adults (over age 18 years) living in private households or 55,000 people and 4.9% or 12,500 people living in long-term residential care facilities (Statistics Canada, 2015).

PD is characterized by a progressive loss of dopaminergic neurons in the substantia nigra pars compacta area of the brain (Brug, Singleton, Gasser, & Lewis, 2015). The dopaminergic cell loss in the substantia nigra pars compacta is associated with decreased striatal dopamine concentrations, which results in disruption of the basal ganglia-thalamocortical motor circuit. Specifically, abnormally high neural discharge from the basal ganglia motor output nuclei, substantia nigra pars reticulate, and internal segment of the globus pallidus are believed to cause hypokinesia or reduced force and range of movement due to increased inhibition of motor cortical regions (Abbruzzese & Berardelli, 2003). This disruption enables diagnosis of PD to be primarily based on observable clinical signs. The major motor features of the disease include the symptoms of rest tremor (3-5Hz frequency), rigidity (increased, sustained muscle tone), akinesia (reduced number of spontaneous movements), bradykinesia (slowed movements), hypokinesia (reduced range of movements), and postural instability. Other secondary motor symptoms may be observed as well such as hypomimia, dysarthria, dysphagia, micrographia, shuffling gait, freezing of gait, festination, and dystonia. In addition, non-motor symptoms of the disease include autonomic dysfunction, cognitive impairment, sleep disorders, and sensory abnormalities (anosmia, ageusia, pain, paresthesias) (Duffy, 2013; Hobson, 2003; Jankovic, 2008).

Subtypes of PD have been identified with a classification based on age of onset and motor symptoms (Ma, Chan, Gu, Li, & Feng, 2015). Age based subdivisions include juvenile, young, and late onset groups, while motor subtypes include hypokinetic rigid, tremor dominant, and postural instability-gait disorders (Ma et al., 2015). Although the specific etiology of PD is unknown, several environmental risk factors have been identified and some studies show that genetic factors may be contributing, particularly in patients with young-onset PD (Olanow & Tatton, 1999). Autosomal dominant forms of the disease have been identified since the late 1990's, and today over a dozen genes (including *SNCA*, *PARK2*, *PINK1*, and *LRRK2*) are implicated in familial PD and other syndromes where Parkinsonism is a prominent symptom (Brug et al., 2015).

Treatments used to help control PD symptoms include drug therapy, behavioural therapy, and surgery (deep brain stimulation, ablation) (Parkinson Society Canada, 2003). The total cost for PD in Canada, including hospital care, drug therapy, and long-term disability care, is around $558 million annually (Parkinson Society Canada, 2003).

1.1 Motor Deficit vs. Sensory/Somatosensory Deficit in PD

Many researchers believe that the degenerative process of PD is primarily based on degeneration of the motor system(s) (Duffy, 2013). There is, however increasing evidence that non-motor symptoms including sensory or sensorimotor dysfunction may be impacted as well (Abbruzzese & Berardelli, 2003; Patel, Jankovic, & Hallett, 2014; Schneider, Diamond, & Markham, 1986; Tatton, Eastman, Bedingham, Verrier, & Bruce, 1984). The broad range of motor and non-motor symptoms can be attributed to striatal dopamine deficiency along with central and peripheral dopaminergic and non-dopaminergic pathways (Patel et al., 2014). Studies by Braak, Ghebremedhin, Rüb, Bratzke, and Del Tredici (2004) have found neuropathological alterations outside of the substantia nigra region which correlate with non-motor symptoms such as olfactory dysfunction as well as autonomic and sleep disturbances.

Researchers have examined sensory and somatosensory deficits in PD as a component of a largely motor-based disease. Visual-spatial and visual postural deficits have been documented in PD (Boller et al., 1984; Bronstein, Hood, & Gresty, 1990). Deficiencies in somatosensory tactile and proprioceptive mechanisms primarily occur early in the disease progression (Conte, Khan, Defazio, Rothwell, & Berardelli, 2013; Govil et al., 2013). It may be assumed that sensory abnormalities are mediated by the basal ganglia circuitry. Some authors purport that sensory deficiencies underlie several of the motor symptoms (Govil et al., 2013). In addition, it is important to note that the basal ganglia are considered responsible for gating sensory input for motor control (Kaji, 2001; Kaji, Urushihara, Murase, Shimazu, & Goto, 2005).

1.2 Sensorimotor Integration Deficit in PD

Voluntary movements depend heavily on peripheral sensory feedback. PD-related bradykinesia and rigidity have been hypothesized as being related to abnormal processing of mechanocreceptor sensory inputs for movement production (Tatton et al., 1984). Therefore, it may be possible to describe PD as involving a sensorimotor integration deficit rather than a motor deficit with some sensory abnormalities. Sensorimotor integration refers to a process by which peripheral sensory pathways convey information to cortical motor pathways and this information is then integrated by the central nervous system in order to complete motor program execution (Abbruzzese & Berardelli, 2003). A deficit in sensorimotor integration involves abnormal processing of the sensory information (afferent input or neural response to input) for motor execution.

Several studies have explored the sensorimotor integration deficit involved in PD movement control (Almeida et al., 2005; Bronstein, Hood, & Gretsy, 1990; Klockgether & Dichgans, 1994; Rinalduzzi et al., 2015). Methods of evaluating sensorimotor integration processes include manipulating sensory feedback, such as visual feedback. Individuals with PD have displayed overreliance on visual information during movement towards a target and were more affected by absent visual feedback (walking in complete darkness) than controls (Almeida et al., 2005). Klockgether and Dichgans (1994) conducted a study on upper limb movements and found that when PD participants were blocked from seeing their moving hand, movement accuracy (undershoot) and speed were more severely impacted compared to controls. In addition, during postural stability tasks, PD participants have shown overreliance on visual information causing instability which control subjects were able to attenuate, indicating a potential sensorimotor integration deficit (Bronstein et al., 1990; Rinalduzzi et al., 2015). A study by Teulings, Contreras-Vidal, Stelmach, and Adler (2002) also found overreliance of visual feedback by individuals with PD in a writing task. This finding was dissimilar to healthy controls that would update their original prediction using the manipulated visual feedback and made corrections to their handwriting movements in the expected/opposite direction to the perturbed error (Teulings et al., 2002).

1.3 Speech in PD

The speech characteristics of the PD population have been classified as hypokinetic dysarthria (HKD) due to the hypokinetic symptoms of the speech system (reduced force and amplitude of movement). Hypophonia or low speech intensity has been found to be the most common speech symptom experienced by individuals with PD, across age and disease duration (Adams & Dykstra, 2009; Darley, Aronson, & Brown, 1969; Duffy, 2013; Logemann et al., 1978; Wertheimer et al., 2014). The term hypophonia has been in use since at least 1930, when Kennedy used it to describe the "whispered or near-whispered" speech of certain individuals with mental illness (1930). Many allied health professionals as well as researchers have used this term over the years, however the definition of the term has varied. It has been used to describe a reduced frequency of vocalizations, a reduced speech volume, and breathy phonation (Brin, Blitzer, Fahn, & Lovelace, 1989; Langston, Forno, Rebert, & Irwin, 1984; Meissner, Sapir, Kokmen, Stein, & Report, 1987). More recently it has been used to describe overall reduced speech loudness (Duffy, 2013).

Despite differences in methods of measurement of intensity, researchers have found a significant reduction in average speech intensity in participants with PD relative to healthy control speakers (Adams et al., 2006; Adams et al., 2008; Adams, Winnell, & Jog, 2010; Ho, Bradshaw, Iansek, & Alfredson, 1999; Ho, Iansek, & Bradshaw, 2000;

Rosen et al., 2006). On average, individuals with PD have reduced average speech intensity levels (2-4dB lower) compared to age-matched, healthy control speakers (Adams, Haralabous, Dykstra, Abrams, & Jog, 2005; Clark, Adams, Dykstra, & Moodie, 2014; Fox & Ramig, 1997; Ho et al., 1999). Darley et al. (1969) described speech loudness as a factor that impacts speech intelligibility. Speech intelligibility, when rated as low by either a listener or the speaker, can have negative consequences for a number of aspects of life. This could include overall quality of life, activity, and participation (Dykstra, Hakel, & Adams, 2007). Reduced loudness and loudness variability (monoloudness) have been implicated in reduced overall quality of life, withdrawal from social interactions, and decreased participation (Miller, Noble, Jones, & Burn, 2006).

The relationship between the major motor symptoms of PD and speech symptoms are unclear, suggesting that basal ganglia involvement in speech may be unique and more complex. It has been argued that mechanisms of speech control are fundamentally different than other motor movements, as a result of differences in neural control and muscle fiber makeup (Kent, 2004). Consistent with this, Braak and colleagues (2004) specified that striatal dopamine depletion likely occurs relatively later in disease progression, whereas speech symptoms such as hypophonia tends to be one of the earliest symptoms. This may be potentially related to vagal and glossopharyngeal nerve involvement (non-dopaminergic neurotransmission) which is affected in early stages of PD progression (Braak et al., 2004). The specific pathological mechanism causing speech impairment in PD is unclear, however it is possible that sensory or sensorimotor integration deficits constitute this aspect of PD.

With regards to PD speech and the oral-motor system, there has been some evidence for the implication of sensorimotor deficits on motor performance. A review by Sapir (2014) concluded that PD related speech impairment is attributed to multiple factors including sensory processing. A study on oral-lingual-facial sensory and motor functions by Schneider, Diamond, and Markham (1986) found that individuals with PD were more impaired in tests of sensory function and sensorimotor integration compared to controls. Specifically, they found significant impairment in jaw proprioception, tactile localization on the tongue, gums and teeth, as well as difficulty performing targeted head movements on the basis of tactile sensory information despite having adequate motor control of head movement (Schneider et al., 1986). In addition, Hammer and Barlow (2010) found reduced vocal tract somatosensory feedback in PD, and Hegland, Troche, and Brandimore (2019) found reduced perception of general airway somatosensation in PD. These findings are important, as the vocal tract and respiratory system are integral for speech production. Overall, PD speech has shown to be linked to multiple factors including motor planning, initiation, as well as scaling, sensory processing, vigilance, and even depression and cognitive-linguistic processing (Sapir, 2014).

1.4 Auditory Feedback for Speech Intensity Regulation in PD

Sensorimotor integration deficits for the regulation of speech intensity may involve abnormalities in auditory perception during speaking tasks. The importance of auditory processing for speech is evident during child development when acoustic input heavily influences the speech patterns of pre-lingual children. Further evidence of the importance of auditory information for speech production includes research suggesting that the low speech intelligibility of hearing-impaired speakers is a result of the of auditory signal perception impairment. In addition, this is described in studies of post-lingually deafened individuals who present with abnormalities in the loudness as well as pitch and rate of speech (Waldstein, 1990).

Auditory-related dysfunction has been evaluated in PD and may be caused by loss of dopaminergic neurons in the basal ganglia and subsequent projections to the inferior colliculus, medial geniculate nucleus, and temporal cortex. Thus, in PD there may be inefficient cortical control of the auditory system. In fact, auditory evoked potentials (measured using EEG) have been demonstrated as abnormal in PD participants both “on” and “off” medication suggestive of disrupted auditory processing in this population (Lukhanina, Kapustina, Berezetskaya, & Karaban, 2009). Abnormal auditory perception is further corroborated by Arnold, Gehrig, Gispert, Seifried, and Kell (2014) who recruited a pre-symptomatic PD group who later went on to develop hypophonia. They found decreased relative suppression of auditory cortex activity (while hearing one’s own voice) compared to healthy controls during overt reading tasks (Arnold, et al., 2014). The impact of increased activity in the auditory cortex during the reading task may translate to abnormal perception of self-produced speech, however this hypothesis is speculative. In addition, they found hypo-connectivity between the left dorsal premotor cortex and the left auditory cortex, suggestive of a dysfunctional sensorimotor integration function in PD (Arnold et al., 2014). A more detailed discussion of neural networks related to auditory feedback is provided in Section 1.7.

Lower level auditory-perceptual processing deficits such as increased hearing thresholds, abnormal acoustic reflex activity, and abnormal auditory brainstem responses have been observed in PD populations, however only inconsistently (Lai, Liao, Lin, Lin, & Sung, 2014; Murofushi, Yamane, & Osanai, 1992; Vitale et al., 2012; Yýlmaz et al., 2009). The focus on higher level auditory processing of speech intensity information is validated based of the observed perceptual deficits in this population.

Intensity regulation is impacted by a variety of external cues or conditions. In typical conversational settings, the speaker must monitor the environment and their own speech intensity levels in order to compensate for such factors as ambient or background noise in their surroundings as well as how near or far their listener is situated. In addition, the speaker must have a method of regulating their speech intensity while simultaneously performing a separate task. In order to do these things, the speaker must be perceptive to their surroundings, make the necessary alterations to their voice, and also have some sort of sensorimotor monitoring process in place to maintain the adjustment. The varied contexts that a speaker experiences necessarily means that processing of additional factors such as distance, communicative intent, and cognitive load are all implicated in the regulation of speech intensity in naturalistic contexts. In addition, abnormal processing of auditory information during speech may involve deficits in loudness perception.

1.4.1 Speech Tasks

Average speech intensity can be obtained across a vowel, sentence, and across a breath group or utterance within speech (Adams et al., 2005; Huber & Darling, 2012; Neel, 2009). The nature of the speech task has an influence on the regulation of speech intensity (Fox & Ramig, 1997; Rosen, Kent, & Duffy, 2005). Quasi-speech tasks include those that do not necessarily represent natural speaking intensity (e.g. vowel prolongation compared to conversational tasks) (Rosen et al., 2005). Junqua, Finckle, and Field (1999) found speech intensity increased more in background noise (Lombard effect) during conversational speech than in a reading task. The effect of speech task on speech intensity regulation is also exemplified by work conducted by Patel and colleagues (2014). These researchers found healthy participants to regulate speech intensity (during perturbed feedback) only in speaking contexts requiring a specific linguistic goal, specifically relating to emphatic stress in a sentence. However, it is possible that suprasegmental and segmental aspects of speech may be controlled by different mechanisms for which auditory feedback plays different roles (Perkell et al., 2007).

Interestingly, PD participants have been shown to lack an automatic adjustment of their speech intensity in conversational samples, unlike healthy controls (Ho et al.,1999). Whereas healthy controls show a tendency to increase the intensity when speaking in conversational tasks, particularly those with added cognitive requirements (speaking about personal experiences), PD participants do not make a similar adjustment (Ho et al., 1999; Winkworth, Davis, Ellis, & Adams 1994). In fact, Moon (2005) found a greater reduction in speech intensity during conversational tasks compared to reading. The content of the message may play a role in intensity adjustments. This includes communicative intent and emotional content; high emotional content may produce wide ranges of speech loudness, which may not exist in emotionally neutral conversation. In addition, it has been hypothesized that the compounded attentional demands associated with a conversation task may have an impact on speech intensity regulation (Adams & Dykstra, 2009).

The observed deficits in speech tasks and speech intensity modulating conditions may be related to difficulties adapting to the social environment. In addition, because PD is associated with dopamine depletion and this is an important neurotransmitter which signals reward expectation in the striatum (Balleine, Delgado, & Hikosaka, 2007; Daniel & Pollmann, 2014), it will be important to examine differences between speaking tasks. Speech tasks may range in terms of the social rewards attributable to each. Therefore, it may be critical to account for this when evaluating speech intensity regulation in PD populations.

1.4.2 Interlocutor Distance

The talker to listener distance, or interlocutor distance can cause the healthy speaker to increase their speech intensity with increasing distance (Cheyne, Kalgaonkar, Clements, & Zurek, 2009; Traunmüller & Eriksson, 2000). We can examine the interlocutor distance slope of the function by comparing changes in interlocutor distances to speech intensity. Researchers have found that PD participants are able to regulate their speech intensity at a similar rate to healthy controls, however PD speech intensity is at an overall reduced level at each distance compared to control speakers (Adams, Winnell, & Jog, 2010; Ho et al., 1999; McCaig, Adams, Dykstra, & Jog, 2015).

The difficulty with modulation of speech intensity for varying interlocutor distances in PD may be related to a difficulty with processing visual distance information. Individuals with PD have displayed overreliance on visual information during distance judgement tasks and underestimations of distance compared to healthy controls (Almeida et al., 2005; Martens, Ellard, & Almeida, 2013). It is also possible that the observed deficits in speech intensity modulating tasks may be related to difficulties adapting to the social environment. Theory of mind (ToM) is a concept related to social cognitive domains. It involves the ability to attribute mental states including beliefs and intentions to others in order to assist with predictions of their mental state and behaviour (Bora, Walterfang, & Velakoulis, 2015). Individuals with PD were found to have significantly impaired ToM compared to controls and this was consistent across a variety of ToM tasks as well as among those in earlier stages of the disease (although significantly less severe compared to later stages) (Bora et al., 2015). However, it may be argued that if distance judgements or ToM were abnormal in the PD population, then the scaling of intensity across distances would be more abnormal. However, the observed deficit in previous research is suggestive of abnormal "gain-setting". The relative influence of gain-setting and visual distance judgements requires further examination.

1.4.3 Background Noise and Lombard Effect

The Lombard effect, first described by Lombard in 1911, is the phenomenon in which a person increases their speech intensity when speaking in a noisy environment. This observation remains consistent across reading and conversational tasks, with several studies providing evidence of healthy speakers increasing their intensity with increasing levels of background noise as well as decreasing their speech intensity once the noise is stopped (Adams et al., 2006; Ho et al., 1999; Lane & Tranel, 1971; Pick, Siegel, Fox, Garber, & Kearney, 1989). Garnier, Henrich and Dubois (2010) also found this effect to be more robust when background noise was played through headphones compared to when played through loudspeakers, however the ecological validity of this should be noted.

Similar to findings from increasing interlocutor distance conditions, individuals with PD-related hypophonia increase their speech intensity as the levels of background noise increase, however their speech is consistently lower than controls across all conditions (Adams & Lang, 1991; Adams et al., 2005). These studies utilized pink and white noise as well as multi-talker background noise. It is interesting to note that these studies found an "overall gain reduction" for speech intensity in PD (Adams et al., 2006; Ho et al., 1999; Iulianella, Adams & Gow, 2008). This is because the PD speakers spoke at a consistently lower intensity despite a fairly typical slope of the regression function (increasing background noise levels produced sequentially increased intensity responses). Adams et al. (2006; 2008) found a gradually decreasing signal-to-noise ratio with increasing background noise, which is suggestive of a perceptual deficit related to the ability to recognize and regulate speech intensity at a level that is appropriate for the listener under these noise conditions.

Type of background noise presented does not appear to have an impact on this Lombard effect in PD, with various intensities of pink noise and music presented in a study by Adams and colleagues (2006). However multi-talker background noise did elicit significantly higher intensity in both PD and controls (Adams et al., 2006). This is in contrast to minimal differences between noise types found by Ho and colleagues (1999). The intensity level of the background noise may also play an important role in the Lombard effect displayed by PD participants. Lane and Tranel (1971) describe the influence of floor and ceiling effects, whereby speech intensity cannot continue to increase despite high levels of background noise and conversely speakers have a lower limit to speech intensity production. PD participants have been hypothesized to have a reduced range of speech intensity production (Clark et al., 2014; De Keyser et al., 2016). Background noise within a range of 50-90dB SPL has been used to depict the Lombard effect in PD and is also comparable to everyday communication contexts (Adams & Lang, 1991; Adams et al., 2005).

1.4.4 Magnitude Production Task

A frequently used method of evaluating autophonic judgement (self-perceived loudness) is through a magnitude production task (MPT). Using this method, the participant initially produces a spoken-stimulus and this production is assigned a value that serves as an anchor or modulus for all subsequent productions. The participant is then asked to produce utterances that are ratios of the initial, anchor production (i.e. two times louder, four times quieter, etc.). This approach is systematic in its method and is based on previous psychometric research. The actual intensity of these autophonic productions is compared to the intended or target intensity values, using regression procedures, and an autophonic loudness function can be obtained (Lane, Catania, & Stevens, 1961).

The MPT requires a scaling of speech intensity and therefore deliberate monitoring of speech production intensity levels via sensory mechanisms. In other words, the MPT involves the relationship between a speaker's perception of their speech loudness and the actual speech intensity produced. Healthy controls are able to produce an autophonic function with a coefficient of 1.17 when obtained by this method (Lane et al., 1961). This means that healthy speakers are able to estimate increases in their own loudness at an almost 1:1 power ratio of the actual sound pressure that they produced.

Previous work by Dromey and Adams (2000) did not find a significant difference between mildly hypophonic PD participants and control subjects when asked to complete this task. It is important to note that these researchers employed a vowel production task (quasi-speech task). In contrast, Clark and colleagues (2014) found that those in the PD group displayed a flatter slope of the loudness function compared to controls when producing a sentence (moderately hypophonic PD participants). This suggests that a scaling ability is present in the PD population, however it also depicts a more restricted range in the perception of loudness.

Using this method there are potential confounds such as the executive functioning ability necessary to remember the previous loudness and working memory to retain each loudness-value match. Therefore, cognitive strength and weakness need to be considered when evaluating the PD population.

1.4.5 Imitation Tasks

Imitation tasks of speech intensity stimuli require processing of an auditory stimulus as well as planning and executing a corresponding speech intensity level. This task allows for a controlled stimulus target, therefore enabling the more precise study of sensorimotor integration for speech intensity. PD participants have been found to produce lower speech intensities in imitation tasks compared to healthy controls (Adams et al., 2006; Clark et al., 2014; De Keyser et al., 2016). Clark and colleagues (2014) and De Keyser and colleauges (2016) found flatter slopes in the imitation function for PD participants (using 60-80dB stimuli in 5dB increments). De Keyser and colleagues (2016) found lowered intensity production levels by the PD participants, however this finding was restricted to the higher intensity imitation condition only (80dB). Adams and colleagues (2006) found 3-4dB differences in intensity (reduced) across the target imitation levels (60, 70, 80dB). However, Adams and colleagues (2006) did not find significantly different slope functions compared to healthy controls. Interestingly, adequate intensity capacity was displayed by PD participants in these studies (as evidenced during maximum intensity tasks), despite the reduced levels produced during the imitation tasks (Adams et al., 2006; Clark et al., 2014; De Keyser et al., 2016). The underestimation and reduced production observed in these studies is suggestive of abnormal processing of sensory information or abnormal sensorimotor integration, or both.

1.4.6 Loudness Perception

Loudness is a psychological characteristic of speech. It is the auditory sensation of speech sounds, which can be ordered on a scale ranging from quiet to loud (American National Standards Association, 1973). The measurement of loudness therefore begins with our perception of loudness itself. It is suggested that our perception and quantification of intensity begins with physical signals that are evaluated by the central nervous system as magnitudes (Warren, 1973).

When estimations of the loudness of speech are examined, it is important to consider who is responsible for the judgement. There are distinct variables to consider if the speaker is making the judgement regarding their self-perceived loudness level (autophonic judgement). This type of judgement presumably accounts for proprioception, acoustic reflex activity, as well as auditory perception through both air and bone conduction (compared to extraphonic judgements of externally generated sounds which are perceived through air conduction alone). Healthy speakers have a magnitude estimation of the loudness function equal to 0.91 when asked to estimate the loudness of their speech using autophonic and extraphonic judgements (Lane et al., 1961).

Anecdotal reports of individuals with PD-related hypophonia describe a lack of awareness of their reduced speech intensity (Dromey & Adams, 2000; Duffy, 1995). A variety of methodologies have been used to explore loudness perception in PD during speaking tasks. Ho and colleagues (2000) found that individuals with PD overestimated their own speech loudness during both immediate and playback conditions when reading and during conversation (using a volume knob to replicate the loudness of the intended stimuli) compared to healthy controls. However, this study did not examine whether this abnormal perception of speech loudness is regarding external stimuli or of self-generated speech loudness only. Conversely, a study by Dromey and Adams (2000) did not find a significant difference between individuals with PD and healthy controls in the perception of speech intensity (loudness estimates, magnitude production task). Kwan and Whitehill (2011) provided evidence of a loudness perception deficit in self-generated speech only. Studies have also provided evidence of abnormal perception of externally generated speech, however to a lesser extent compared to self-generated speech (Clark et al., 2014; Ho et al., 1999; De Keyser et al., 2016). Possible explanations for this difference may relate to the inherent differences between autophonic and extrapohonic types of loudness judgements.

Self-rating scales have also been used in autophonic loudness judgements or loudness perception evaluations. PD participants may be asked to provide a self-rating of their speech intensity by placing a dash along a visual analog scale or an equal appearing interval scale to represent their perception of their speech at a certain point in time. The scale may range from a complete presence of adequate speech volume to complete absence of adequate speech volume. This type of measure has provided evidence that individuals with PD perceive their self-generated speech as significantly more impaired with regards to speech intensity compared to healthy controls (Fox & Ramig, 1997). However, it is conceivable that individuals with PD may not accurately perceive the severity of their hypophonia impairment.

1.5 Altered Auditory Feedback and Speech Intensity Regulation in PD

It is predicted that evidence for a sensorimotor integration deficit hypothesis for speech production would be most apparent during an ongoing speech movement. If during a speech movement one experiences unexpected alterations of the sensory feedback (auditory, visual, proprioceptive) the system should be able to recognize the incongruence from the efference copy (motor plan) and adjust or compensate accordingly. For example, previous literature has described this type of compensatory response by healthy speakers (pitch and formant structure perturbations) as an alteration in speech production in the opposite direction to the perturbation (Purcell & Munhall, 2006; Tourville, Reilly, & Guenther, 2008; Burnett, Freedland, Larson, & Hain, 1998). An alternate method of manipulating auditory feedback is to completely mask perception and evaluate performance in the absence of auditory feedback. In PD populations, it has been suggested that hypophonia may be a result of auditory-motor integration deficits (Adams & Dykstra, 2009). The error correction ability during altered intensity feedback and intensity regulation in the absence of auditory feedback in PD populations may be abnormal and further examination of this abnormality may provide insight into which part of the process is disrupted. Finally, by instructing a speaker to ignore the auditory feedback and measuring their accuracy during this task, it is possible to examine the degree to which the role of auditory feedback for speech intensity regulation is under deliberate and voluntary control.

1.5.1 Altered Intensity Feedback (AIF)

The role of sensorimotor integration for speech intensity regulation can be examined by systematically altering sensory feedback. Abnormal responses during error correction paradigms can indicate a dysfunctional sensorimotor integration system. Altered intensity feedback is proposed to examine this. Findings from this research will help elucidate whether the PD system has under-influence of sensory feedback during speech, or overreliance on sensory feedback. Auditory feedback can be manipulated in a predictable manner (for which Mollaei, Shiller, and Gracco in 2013 found that PD participants responded with reduced magnitude), to explore error-based learning. Conversely, unpredictable manipulations (as will be the focus here) may be used to examine online sensorimotor control.

Perturbation studies involve the rapid response and compensation to a brief (<200ms) perturbation to the speech signal (pitch, formant frequency, duration, intensity, etc.). Healthy participants respond to unexpected brief perturbations of speech intensity by compensating in the opposite direction to the feedback (Bauer, Mittal, Larson, & Hain, 2006; Heinks-Maldonado & Houde, 2005). Studies of auditory perturbation (pitch and formant frequency) have found that PD participants exhibit an abnormal response to sensorimotor integration compared to the control groups (larger magnitude of compensation, longer response peak and end durations) (Chen et al., 2013; Huang et al., 2016; Mollaei et al., 2013, Mollaei, Shiller, Baum, & Gracco, 2016). Similarly, Liu, Wang, Metman, and Larson (2012) found larger response magnitudes to intensity perturbations by PD participants compared to healthy controls.

Altered intensity feedback (AIF) involves the presentation of one’s own speech via headphones for the duration of the utterance. This type of manipulation causes the participant to hear their speech at an altered (increased or decreased) intensity than is actually produced. This results in a healthy speaker adjusting their intensity to speak at a quieter loudness when hearing increased intensity feedback, as a presumed compensatory measure (Ho et al., 1999; Lane, Tranel, & Sisson, 1969; Lane et al., 1961; Siegel & Pick, 1974). Few previous studies have examined responses to AIF in PD. Ho and colleagues (1999) found that individuals with PD failed to adjust their volume in a conversation task, implying a disrupted loudness perception. This study did not evaluate the response of PD participants to decreased intensity feedback. Interestingly, separate results from syllable, reading, and counting tasks depict the PD group responding similarly to controls (Brajot, Shiller, & Gracco, 2016; Coutinho, Oliveira, & Behlau, 2009; Ho et al., 1999), suggestive of a possible task effect. There is a dysfunctional system in PD as evidenced by abnormal responses during error correction tasks. Due to limited previous research, the impact of altered intensity feedback on speech intensity regulation in PD populations requires further exploration.

1.5.2 Complete Masking of Auditory Feedback

Speaking in complete masking noise may also provide evidence of the role of auditory feedback during speech. Researchers determined that vowel space decreased and vowel dispersion measures increased when speaking in a high level masking noise condition (which completely masked auditory feedback) (Lane et al., 2005). The increased dispersion has been related to reduced vowel contrast and therefore results in decreased speech intelligibility of the speaker. Other researchers have provided evidence of minimal changes from background noise conditions to high level masking noise conditions (2dB increase in speech intensity), leading to the hypothesis that speech task may impact production change to a greater degree than noise levels alone (Van Summers, Pisoni, Bernacki, Pedlow, & Stokes, 1988). This may relate to Lindblom's notion of economy of effort (1990), whereby it is possible that when speech is perceived through the auditory system, speakers are better able to control and finely tune speech intensity for the speaking task, however in the presence of a degraded sound environment such as complete masking noise, intensity compensations or adjustments for the speech task may be lowered in priority.

Researchers have examined differences between pre-lingually deafened and post-lingually deafened individuals and have found that post-lingually deafened speakers rely less on auditory feedback monitoring due to already established speech sound control (internal representations). In addition, Black (1951) found linear increases in speech intensity with exposure to noise-induced hearing loss. However, the impact on PD speakers is worth investigating as it is unpredictable how perceptual mechanisms are impacted in this disease. In addition, the specific impact of absent auditory feedback on speech intensity regulation in the PD population is yet to be examined. If unable to utilize auditory feedback mechanisms will individuals with PD be capable of regulating speech intensity through previously learned speech sound control? Will these individuals be more severely impacted compared to neurologically healthy speakers? To our knowledge, no study to date has explored the impact of completely masked auditory feedback on speech intensity regulation in PD populations.

1.5.3 Instruction to Ignore Auditory Feedback

Munhall, MacDonald, Byrne, and Johnsrude (2009) found that healthy subjects compensated for altered feedback whether they were provided instructions to ignore the feedback or not suggesting absence of conscious awareness of these compensations. The degree to which the system relies on sensory feedback will also depend on the reliability of the source (Shadmehr, Smith, & Krakauer, 2010; Sober & Sabes, 2005). Due to the progressive nature of PD, some aspects of sensory processing may be weighted as less reliable (unpredictable) or more reliable (predictable). For example, it is possible that the acoustic reflex and/or auditory nerve are compromising the integrity of the auditory system (Yylmaz et al., 2009; Gawel, Das, Vincent, & Rose, 1981). This may result in under-reliance of auditory information during speaking tasks in individuals with PD. Lametti, Nasir, and Ostry (2012) found that by applying two perturbations simultaneously (one auditory one somatosensory; pitch perturbations and robotic arm making subtle jaw displacements), subjects preferentially rely on one or the other. In other words, the more they compensated for one perturbation, the less they compensated for the other. Therefore, it may be possible to assume that with altered auditory feedback alone, there is heavy reliance on jaw/facial sensory input as this may be the more reliable source. Similarly, Larson, Altman, Liu, and Hain (2008) found larger compensatory pitch responses when anaesthetic spray was applied to the vocal folds presumably causing uncertainty from the somatosensory feedback. It is unclear how participants would respond if given the explicit instruction to avoid using altered feedback ("this auditory signal is incorrect").

Evidence from a study using instructions to increase speech intensity suggests the importance of explicit instruction for successful speech regulation (Ho et al., 1999). However, Pick and colleagues (1989) found that conditions, during which participants were asked to inhibit the Lombard response, resulted in unsuccessful attempts. Instruction to ignore altered intensity feedback during speech tasks could help explore the ability to internally regulate speech perception for production purposes (regulation of the feedback system).

1.6 Auditory Feedback for Speech Intensity Regulation in PD: Theoretical Models

Several theoretical models have been proposed and may serve as a basis from which to understand how sensorimotor processes may be functioning for speech intensity control in PD populations. Feedforward processes do not incorporate sensory feedback as having a primary cue during a movement. This model is used during situations when stable feedback is not possible (e.g. in background noise, delayed feedback due to synaptic and processing delays, masking noise). The BG-SMA (basal ganglia-supplementary motor area) circuit is thought to play a primary role in feedforward control (Cunnington, Bradshaw, & Iansek,1996; Nixon and Passingham, 1998). This process may be compromised in PD, due to loss of dopaminergic neurons in the BG (Haslinger et al., 2001). State feedback control models (SFC) postulate an online feedback control which comes from internally maintained representations through which an internal model makes estimates of a motor movement based on previously learned associations. Therefore, the SFC involves both an internal forward model combined with actual feedback used to train over time. The feedback control theory of speech motor control (Fairbanks, 1954 adapted from Wiener, 1948) suggests that motor movements are a sequence of desired sensory outcomes. This is described in more detail below.

The existing literature suggests that speech production and perception rely on a large network of interconnected brain regions, rather than independent areas (Baum & Pell, 1999; Friederici & Alter, 2004; Golfinopolous, Tourville, & Guenther, 2010; Hickok & Poeppel, 2000; Pulvermuller, 2005). This requires attention towards a more holistic model of perception and production network deficits. The Directions into Velocities of Articulators (DIVA) theoretical model of speech motor movements provides a framework for these processes (Guenther, 1994). The motor command for speech is first encoded and sent to the associated muscles. A copy of this motor command (efference copy) is also processed and is used to predict the consequences of the action (Bays, Wolpert, & Flanagan, 2005; Voss, Bays, Rothwell, & Wolpert, 2007; Wolpert & Ghahramani, 2000). With regards to speech intensity regulation, this efference copy must also incorporate or predict high amounts of variability in the environment such as background noise and how near of far the listener is situated. It is possible that hypophonia, or reduced speech intensity, is caused by a reduced efference copy or an abnormal prediction, however based on the limited available evidence there is also potential for alternative hypothesis related to deficits in the processing of auditory feedback.

Once the movement is taking place, there is additional information that needs to be processed and integrated in order for the speaker to maintain the movement based on the prediction. This additional sensory information is also needed to make updates to the movement. Therefore, a functional system requires both a prediction (efference copy) and sensory feedback (updates during movement). Fairbanks (1954) described the idea of a “comparator” which subtracts sensory feedback from an internally generated target to create an error signal during altered feedback. If components of this process are disrupted in PD, this could lead to hypophonia. Another potential explanation for reduced speech intensity may be during the movement itself. Some aspect of sensory processing may be disrupted; causing the individual to perceive these updated signals as increased, thereby reducing their speech intensity as a compensatory measure.

A healthy system requires both a functional/accurate prediction of the motor output (forward model) as well as sensory feedback for monitoring and maintenance purposes (feedforward processes). The relative amount of involvement of each process may shift form task-to-task and context-to-context. The current study proposes to examine speech intensity regulation in a wide range of tasks and contexts and with altered auditory feedback conditions, therefore providing the opportunity to evaluate the control mechanisms that may be disrupted in the PD population.

1.7 Neural Pathways for Auditory Feedback and Speech Intensity Regulation

It has been suggested that the neural control of auditory feedback involves numerous structures and pathways. A detailed exploration of these pathways is beyond the scope of this thesis, however, in order to provide a description of the possible mechanisms that underlie the processes in the current study, a brief overview of relevant neural structures is provided.

The auditory and speech motor control systems have anatomical connections through the pontine nuclei and cerebellum (Glickstein & Mitchell, 1997), putamen, globus pallidus, thalamus (Alexander & Crutcher, 1990; Yeterian & Pandya, 1998), and what is known as the dorsal auditory stream involving the posterior superior temporal gyrus (STG) and the superior parietal temporal area (Spt) (Buchsbaum, Hickok, & Humphries, 2001; Hickok, Buchsbaum, Humphries, & Muftuler, 2003; Zheng, Munhall, & Johnsrude, 2010). This dorsal auditory stream is thought to be specifically involved in feedback processing related to discrete speech production-related perceptual judgments, however lesion studies have examined the role of these structures in processing of phonological factors only (Baker, Blumstein, & Goodglass, 1981; Miceli, Gainotti, Caltagirone, & Masullo 1980). It is possible that this type of feedback monitoring is related to speech intensity, however to our knowledge no previous studies have been conducted to examine this speech characteristic. Importantly, these studies suggest multiple possible auditory-speech motor pathways including transmission of information through subcortical structures that may be implicated in PD-related hypokinetic dysarthria.

Functional imaging studies have also been conducted and areas in the brain that have been shown to be more active during speaking (versus listening), include a number of bilateral motor areas including the anterior cingulate cortex (ACC), supplementary motor area (SMA), anterior insula, and dorsal motor cortex (Christoffels, Formisano, & Schiller, 2007; van de Ven, Esposito, & Christoffels, 2009). Subcortical structures such as the pons, thalamus, and basal ganglia were also shown to be active while speaking (Christoffels et al., 2007; van de Ven et al., 2009). Jurgens (2002) similarly suggested the ACC may be involved in the control of voluntary intonations during speech, the periaqueductal gray (PAG) is involved in modulating intensity, while the brainstem reticular formation (RF) is involved in execution of these structures' pathways.

Speaking and regulating speech intensity in background noise presents potentially different challenges as they relate to signal-to-noise ratios of speech. Callan, Jones, Callan, and Akahane-Yamada (2004) proposed that the ventral pre-motor cortex (PMC) including the posterior part of Broca's area (pars opercularis) are involved in speech perception in noise. However, it is important to note that the degree to which these structures are involved in the perception of one's own voice in noise is unclear as the Callan and colleagues (2004) study involved the perception of speech recordings.

Speaking-induced suppression (SIS) has been observed in the auditory cortex (AC) during self-produced speech such that the activity in the AC is reduced compared to when externally-produced speech is played to a participant (Curio, Neuloh, Numminen, Jousmaki, & Hari, 2000; Greenlee et al. 2011; Houde & Jordan, 2002). Some functional imaging research has focused on neural activity in the context of altered feedback and speech compensations. Interestingly, the SIS phenomenon does not occur when the participant is presented with altered auditory feedback (Behroozmand, Karvelis, Liu, & Larson, 2009, Chang, Niziolek, Knight, Nagarajan, & Houde, 2013, Eliades & Wang, 2008, Greenlee et al., 2011; Houde & Jordan 2002) suggesting that although the auditory cortex functions to suppress function with expected auditory feedback, once there is a mismatch with this expectation, the auditory cortex is once again primed. Studies have found the superior temporal gyrus (STG) (Fu et al., 2006; Tourville et al. 2008; Parkinson et al. 2012; Zheng et al. 2010), and ventral supramarginal gyrus (vSMG) (Tourville et al. 2008; Toyomura et al., 2007) to be active during altered auditory feedback. Tourville and colleagues (2008) also found activation in superior cerebellum, ventral thalamus and anterior striatum, with the additional regions of bilateral superior cerebellar cortex, medial parietal-occipital cortex and right lateralized inferior cerebellar cortex active during altered pitch feedback. Thus, complex sensory-motor networks are involved in speech production with altered auditory feedback and sensory activation of motor control areas may be responsible for the compensation of erred feedback.

Some auditory cortical areas have been observed to increase in activity, known as a speech perturbation response enhancement (SPRE), with altered auditory feedback (Behroozmand et al. 2009; Zheng et al. 2010). Importantly however, these studies focused on examining aspects of speech other than intensity (e.g. pitch) and often in the context of syllables rather than full utterances. The implications of these differences are potentially important and therefore further research is required to better understand the neural structures involved in altered/unaltered intensity feedback. In addition, some studies have found the mid-to-posterior STG to be more active when auditory feedback was completely masked (Christoffels et al. 2007; van de Ven et al. 2009), highlighting the importance of the STG in auditory processing of self-generated speech.

Some studies have examined the areas related to auditory perception and speech-motor areas in the PD population. New and colleagues (2015) found reduced resting state connectivity between the thalamus and putamen with cortical motor areas including the STG. Rektorova and colleagues (2012) showed that as speech intensity increased during an overt reading task in the PD participants, the magnitude of connectivity between the PAG and the right posterior STG increased. It is possible that this is related to compensatory mechanisms or a result of dopaminergic therapy (Rektorova et al., 2012). This evidence suggests possible connectivity issues between key structures involved in auditory perception for speech regulation and provides avenues for future research as it relates to neural structures related to auditory-speech-motor control.

1.8 Rationale for Proposed Study

Researchers have studied the relationship between speech perception and production in PD-related hypophonia. It is possible that auditory perception for speech may be impaired in this population. Despite the work that has been conducted on speech intensity perception and production, there is a paucity of literature that has addressed this issue in the context of the range of communicative situations and speech tasks experienced by these individuals. Using the altered intensity feedback paradigm and complete masking procedure, changes in speech production and perception can be measured and this enables the study of how the speech motor system responds to auditory alterations. The proposed novel approach will examine the impact of AIF during multiple speaking tasks with and without instructions to maintain constant speech intensity. Also, examination of a range of intensity feedback distortions including reduced intensity feedback through an AIF task will help determine upward scaling abilities in PD, as the Ho and colleagues (1999) work was restricted to increased intensity of auditory feedback.

There is a paucity of research examining responses to AIF during various speech tasks. It is possible that the sensorimotor integration for speech intensity regulation is differently influenced by both auditory perception and the nature of the communicative goal. No study to date has explored the differences between quasi-speech and standard speech tasks during altered intensity feedback and its impact on speech intensity regulation in PD populations. The influence of type of speech task will be important to evaluate in PD populations as cognitive influences impact communication in daily life and therefore including ecologically valid tasks is critical.

Interestingly, the relationship between abnormal perceptual deficits and abnormal Lombard responses has not been examined in previous studies of PD participants. In particular, the response to AIF in background noise by PD participants will be important to examine in order to understand the regulation of speech intensity in ecologically valid communicative contexts. No study to date has explored the impact of background noise during altered intensity feedback and its impact on speech intensity regulation in PD populations. Differences in speech intensity regulation in background noise between PD and controls may indicate abnormal internal representations of loudness.

To date, few studies have explored the magnitude production task in PD participants, and no studies have explored the MPT under AIF conditions. Due to the auditory perception component inherent in the MPT, the manipulation of auditory feedback will necessarily influence the task. Autophonic loudness taps into deliberate self-regulation of speech intensity (based on any mechanism; auditory, cognitive, proprioceptive). The examination of magnitude production scaling during auditory feedback manipulations, may uncover new information about the relative importance of internal estimates of loudness (autophonic) and external intensity feedback processes in the intensity regulation problems in PD. In addition, the imitation task completed during AIF is yet to be examined. The role of auditory feedback for regulation of self-produced speech intensity during an imitation task is important to explore as this may provide information about the processing of an externally generated auditory stimulus as well as planning and executing a corresponding speech intensity level. With regards to communication context the effect of interlocutor distance on speech intensity during AIF has not been examined. This task will indicate the role that distance judgement and auditory feedback plays in speech intensity regulation.

Loudness perception plays a potentially critical role for understanding the disrupted speech intensity regulation in PD populations. Although loudness perception in PD has been studied during speech tasks, perception of loudness during altered feedback conditions has yet to be evaluated. AIF provides the opportunity for systematic manipulations of auditory feedback. Evaluating loudness perception during these manipulations of auditory feedback will enable the study of potential perceptual dysfunction in PD populations. In addition, the perceptual judgement of loudness while in background noise conditions will provide new information about the accuracy of loudness perception in noise in PD.

Loudness perception may be an integral component to understanding the sensorimotor integration of speech intensity in PD populations. There is a gap in the literature with regards to loudness perception during AIF. Similarly, there is a gap in the literature with regards to loudness perception in background noise, which is an ecologically valid situation during which individuals need to make loudness judgements of their speech intensity. Loudness perception tasks completed in background noise will be important for understanding the capacity to direct attention to discrete components of the acoustic environment (discerning background noise levels from self-produced intensity levels).

The altered intensity feedback paradigm uncovers the response to intensity shift/error correction. The focus is on examining the role that auditory sensory feedback plays in intensity control during speech tasks including socially driven speech tasks and naturalistic speaking contexts such as background noise that are known to impact speech intensity.

1.9 Objectives

The purpose of this study was to examine the role of auditory feedback for sensorimotor control of speech intensity regulation in PD.

The following seventeen specific objectives were examined in this study:

*Objective 1. T*o examine the effect of altered intensity feedback during speech production in participants with PD and controls.

Objective 2. To examine the effect of different speech tasks on speech intensity and the response to altered intensity feedback in PD and control participants.

Objective 3. To examine the effects of background noise conditions on speech intensity and the response to altered intensity feedback in PD and control participants.

Objective 4. To examine the effect of a magnitude production task on speech intensity and the response to altered intensity feedback in PD and control participants.

Objective 5. To examine the effect of background noise on the response to the magnitude production task in PD and control participants.

Objective 6. To examine the effect of an imitation task on speech intensity and the response to altered intensity feedback in PD and control participants.

Objective 7. To examine the effect of background noise on the response to the imitation task in PD and control participants.

Objective 8. To examine the effect of complete masking noise and speech tasks on speech intensity in participants with PD and controls.

Objective 9. To examine the effect of complete masking noise and magnitude production task conditions on speech intensity in participants with PD and controls.

Objective 10. To examine the effect of complete masking noise on speech intensity and performance on the intensity imitation task in participants with PD and controls.

Objective 11. To examine the effect of instructions to ignore auditory feedback on speech intensity and the response to altered intensity feedback in PD and control participants.

Objective 12. To examine the effect of the instruction conditions on the response to background noise conditions in PD and control participants.

Objective 13. To examine the self-loudness perception ratings of speech intensity in the context of the magnitude production task and the response to altered intensity feedback in PD and control participants.

Objective 14. To examine the effect of background noise on self-loudness perception ratings in the magnitude production task by PD and control participants.

Objective 15. To examine the self-loudness perception ratings of speech intensity in the context of the instructions to ignore auditory feedback and the response to altered intensity feedback in PD and control participants.

Objective 16. To examine the effect of background noise on loudness perception ratings in the instruction to ignore conditions by PD and control participants.

Objective 17. To examine the self-loudness perception ratings of speech intensity in the context of complete masking noise in the magnitude production task by PD and control participants.

Chapter 2: Methods

2.1 Participants

Twenty-seven individuals with PD and twenty-six neurologically healthy control (HC) participants were recruited for the study. Data from twenty-six PD participants (19 male and 7 female; 69.38 ± 6.38 years) and twenty-four control subjects (8 male and 16 female; 73.29 ± 5.98 years) were analyzed following the exclusion of 1 PD participant due his inability to complete the full study protocol for scheduling reasons, exclusion of 1 control participant due to a technical issue with the audio recording, and another control participant not meeting eligibility criteria for no prior speech disorder. There was no significant difference in age between the PD and HC groups (t(48)=-1.517, p=.136). PD participants were recruited from patients seen by a movement disorder neurologist, Dr. Mandar Jog, and were diagnosed by him as having PD and some degree of hypophonia. Control participants were recruited from the Research Retirement Association in London as well as the Western University Alumni Association. Participants had no other speech/language impairments besides those resulting from a diagnosis of Parkinson’s disease. PD participants were stabilized on their anti-parkinsonian medication and were tested approximately one hour after taking their regularly scheduled dose. The mean disease duration since diagnosis was 8.08 ± 5.09 years. Cognition was assessed using the Montreal Cognitive Assessment (MOCA) and in the normal range (>22). Both individuals with PD and control participants passed a binaural hearing screening with thresholds of 40dB hearing level at .25, .5, 1, and 2kHz frequencies. All participants provided written consent for participation in the study and the research protocol was approved by the Human Subjects Research Ethics Board (HSREB) (Western University Ethics (WUE) No. 109016). PD patient demographics are reported in Table 1.

Table 1. ***PD patient demographic information.***

Participant	Gender	Age	PD Duration	Hypophonia Severity	UPDRS III
PD 01	F	68	7	mild	18
PD 02	M	71	13	moderate	NA
PD 03	M	78	NA	moderate	NA
PD 04	M	69	6	moderate	36
PD 05	M	80	14	moderate	35
PD 06	M	69	12	mild	25
PD 07	M	75	4	moderate	NA
PD 08	F	56	3	moderate	NA
PD 09	M	66	10	mild	19
PD 10	M	83	9	moderate	NA
PD 11	M	68	3.5	mild	11
PD 12	M	70	13	mild	21
PD 13	M	71	5	mod-severe	34
PD 14	M	74	2	mild-mod	27
PD 15	M	69	10	mild	17
PD 17	M	74	2.5	mild	20
PD 18	M	63	6	mild	35.5
PD 19	M	78	3	mild	26
PD 20	M	73	7	mild	25.5
PD 21	M	63	7	moderate	25.5
PD 22	F	73	25	mild	32
PD 23	F	74	11	mild	17
PD 24	M	72	8	moderate	30
PD 25	F	54	5	mild	20

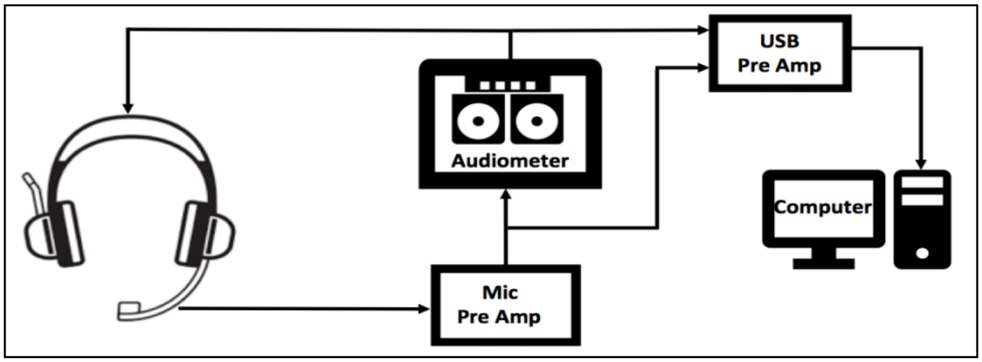
PD 26	F	68	4	moderate	13
PD 27	F	64	12	mild	17

Note. PD = Parkinson's disease; Hypophonia severity = as rated by experimenter; UPDRS III = Unified Parkinson's Disease Rating Scale (Part III: Motor Examination); NA = Data not available

2.2 Apparatus

Participants were seated in an audiometric booth for the duration of the study. Participants were provided with a standard set of audiometric headphones (Telephonics 51OCO17-1) and headset microphone (AKG C520) attached to a preamplifier (M-Audio preamp USB), audiometer (GSI-10, model 1710), and desktop computer. A schematic of the experimental setup is provided in Figure 1. The microphone was placed 6 cm from the midline of the participant's mouth. Calibration of the microphone was established through the use of a sound level meter placed 15 cm (6 inches) from the participant's mouth while they produced three short (<5sec) 'ah' sounds at 70 dBA SPL. The recording module in the Praat software (Boersma & Weenink, 2011) was used to digitize the speech samples at 44.1 kHz and 16 bits. During speech tasks, the audiometer was used to alter the intensity of the participant's speech. The headphone output was calibrated (made equivalent) to the input microphone using speech noise produced by the audiometer and an audio speaker placed 6 cm from the headset microphone. The calibration of the output of the headphones was accomplished with an earphone coupler (Bruel & Kjaer, type 4152) attached to a sound level meter (Bruel & Kjaer, type 2203). Auditory speech stimuli in the imitation task were presented through the same headphones through which participants were presented AIF. The headphones were connected to an audio amplifier that received the calibrated speech stimuli from the audio output of a laptop computer, which played the prerecorded audio (.wav) files. The computer program GoldWave (http://www.goldwave.com) was used to amplify or attenuate the speech audio file and create the target level experimental stimulus files (i.e. 50, 60, 70, 80 dBA SPL). For the measurement of speech intensity in all conditions and tasks, the recorded speech audio files were measured off-line using the acoustic intensity measurement module in the Praat program. Using Praat, long (+250ms) unvoiced segments or pauses were selectively removed and the root mean squared (RMS) intensity contour method was used to obtain the average intensity for each utterance.

Figure 1. ***Schematic representation of the experimental setup.***



2.3 Procedures

The general conditions for this study were presented in the following order: altered intensity feedback, AIF with background noise, complete masking of auditory feedback, and instructions to ignore auditory feedback. The order of all conditions was selected so as to minimize the influence on other tasks of the instruction to “ignore the auditory feedback” in the final condition as well as any potential residual Lombard effect from the background noise conditions. It is important to gather participant responses to altered feedback and communication contexts (i.e. background noise) with minimal knowledge or sensitivity to the production of their speech so as to ensure that the natural response of the auditory feedback system is recorded. The order of specific speech tasks was as follows: (1) conversation (near interlocutor distance), (2) conversation (far interlocutor distance), (3) vowel production, (4) reading at habitual speech intensity, (5) reading 2x louder than habitual, (6) reading 4x louder, (7) reading at maximum loudness, (8) imitation of 50dB stimuli, (9) imitation of 60dB stimuli, (10) imitation of 70dB stimuli, (11) imitation of 80dB stimuli, production of tasks (1-11) in background noise, (12) reading with instruction to ignore auditory feedback, (13) reading with instruction to ignore auditory feedback in background noise, production of all tasks (1-11) in complete masking noise. Several acoustic differences (longer vowel durations, longer voice onset times, etc) have been previously associated with vowel and reading tasks (Brown & Docherty, 1995; Kent, Kent, Rosenbek, Vorperian, & Weismer, 1997), and so to avoid this potential influence on the conversation tasks, the conversation tasks were completed first. The MP and imitation tasks were in sequential order to facilitate success of scaling loudness in this task. Speech sensorimotor adaptation has been suggested in some studies (Houde & Jordan, 1998; Purcell & Munhall, 2006) however the short duration of time our participants will be perceiving altered feedback conditions is not expected to elicit this process.

The study protocol was typically completed in a single session with an average duration of 2.75 hours (range = 2.5-3 hours). Participants were provided 1 rest break approximately half way through the study protocol or as requested (typically only the one break was requested and some participants preferred no break). Due to scheduling reasons, 3 PD participants and 1 control participant completed the study across 2 visits. In each of these cases the second visit was within 3 months (range 1-3 months) following the initial visit.

2.3.1 Altered Intensity Feedback (AIF)

The following speech tasks were completed during AIF: conversation with the experimenter at a close distance (1 meter interlocutor distance), conversation with the experimenter at a far distance (6 meter interlocutor distance), vowel production, sentence reading, magnitude production task, imitation task, and instruction to ignore auditory feedback task. Details of these tasks are below. The randomly altered intensity feedback conditions included 6 conditions; 5, 10 and 15dB reductions in the feedback intensity and 5, 10 and 15dB increases in the feedback intensity.

2.3.2 Speech Tasks

Conversation. The conversation task involved the participant discussing with the experimenter emotionally neutral and cognitively low topics such as family, hobbies, recent vacations, etc.

Vowel Prolongation. The vowel prolongation task involved the sustained phonation of "ah" in a comfortable speaking voice for approximately 3 seconds for each condition.

Sentence Readings. Sentences included randomly selected items from the Sentence Intelligibility Test (SIT) (Yorkston, Beukelman & Tice, 1996) as well as a standard sentence that includes a variety of consonant and vowel sounds that can be useful in the acoustic analysis of PD speech "She saw patty buy two poppies"

(Abeyesekera et al, 2019; Knowles et al., 2018). All sentences were printed on paper for participants to read with the experimenter instruction to “read these sentences to me” in order to encourage reading aloud. The sentence “She saw patty buy two poppies” was used for all further analysis in the current study.

2.3.3 Background Noise

Each of the tasks described above in AIF were completed again in a 65dB background noise condition. Multi-talker background noise was presented to the participant through the same headphones as the AIF.

2.3.4 Magnitude Production Task

The magnitude production task involved reading the sentence “She saw Patty buy two poppies”. Reading of this sentence was performed at 1) a comfortable loudness, 2) with the instruction to read at a loudness that was two times louder than normal speaking loudness, 3) with instruction to read four times louder than normal loudness, and 4) with instruction to read at a maximum loudness.

2.3.5 Imitation Task

Previously recorded speech samples of sentence readings played for participants to listen at different levels of intensity. The four intensity presentations included 50dB, 60dB, 70dB and 80dB SPL. The stimuli were presented through headphones. Participants were then asked to imitate the sentences and specifically the loudness of the speech samples played to them. For the imitation task presented in 65dB background noise, the speech stimuli were presented in no noise and participants were presented with the background noise during their production of the imitation only.

2.3.6 Complete Masking Noise

The following tasks were completed during complete masking noise (same as the above speech tasks with AIF): conversation with the experimenter at a close distance (1 meter interlocutor distance), conversation with the experimenter at a far distance (6 meter interlocutor distance), vowel prolongation, sentence reading, magnitude production task, and imitation task. During speech production the participants were presented with higher intensity multi-talker background noise (100dB SPL) so as to completely mask auditory perception of their own speech.

2.3.7 Instruction to Ignore Auditory Feedback Task

This task involved reading sentences from the sentence-reading task described above, however they were asked to ignore the auditory feedback. This was described to participants as an attempt to ignore the feedback coming through the headphones and instead maintain a constant loudness, as the experimenter would hear it, since the experimenter could not hear what was being transmitted through the headphones.

2.3.8 Loudness Perception

Participants were asked to rate the loudness of their speech during 3 of the 7 altered intensity feedback conditions (no feedback, 10dB reduction and 10dB increase), in no noise and in 65dB background noise, in the magnitude production task and instruction to ignore auditory feedback tasks. Participants were also asked to rate the loudness of their speech while completing the MP task in the complete masking noise condition. Rating data were collected using a visual analogue scale (VAS). Participants rated their self-perceived loudness by placing a dash along a line (endpoints labeled low loudness and high loudness).

2.4 Statistical Analysis

2.4.1 Sample Size Determination and Power Calculation

G*Power v3 (Faul, Erdfelder, Lang, & Buchner, 2007) was used to complete the a priori estimated sample size for a comparison involving independent means. The power calculations were based on results from two studies of speech intensity in PD. The Adams and colleagues (2010) study was used to determine effect sizes for PD versus control comparisons, interlocutor distance comparisons, and noise comparisons. The resulting effect sizes ranged from 1.14 to .76. In addition, the Clark and colleagues (2014) study was used to obtain effect size estimates for PD versus control comparisons obtained during intensity imitation tasks and intensity related magnitude production tasks. The resulting effect sizes ranged from 1.71 to .75. Given that the estimated effect size of .75 was the lowest estimate, this value was selected as the most conservative estimate for performing the power analysis for the current study.

This analysis indicated that a sample size of 46 (PD=23; controls=23) would be required to detect a significant effect with an effect size of .75, a power of .80 and an alpha of .05. In order to ensure that this minimum level of power was achieved, an additional 6 participants (3 per group) were recruited in the study although some were subsequently dropped for reasons discussed in detail in Section 2.1. Using the actual sample size that was finally obtained in the present study (PD=26; control=24), a post-hoc power analysis was performed using an effect size of .75, and alpha of .05. This post-hoc analysis provided a power estimate of .83.

2.4.2 Statistical Analysis for Altered Intensity Feedback

All statistical tests were conducted using IBM SPSS Version 21.0 (IBM, 2011). The speech intensity responses to AIF in the PD and control groups were analyzed using two-way repeated measures ANOVA with group (PD vs. healthy controls) as the between-subjects factor, and AIF level (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB) as the within-subjects factor. Any significant interactions lead to post-hoc comparisons. Linear regression analysis was completed in order to examine the relationship between the levels of speech intensity that were produced for each level of altered intensity feedback. A t-test was used to examine the average AIF slopes for the PD and control groups.

To examine the effect of speech tasks on speech intensity and the response to AIF, a two-way ANOVA with speech task (conversation near, conversation far, reading sentences, vowel prolongation) and group (PD vs. healthy controls) was first used to examine the effects of task on speech intensity in the PD and control groups. A three-way repeated measures ANOVA was then used to analyze the effect of speech tasks on the AIF conditions in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and speech task (conversation near, conversation far, reading sentences, vowel prolongation) and AIF level (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB), as within-subjects factors. Any significant interactions lead to post-hoc comparisons. Linear regression analysis was completed in order to examine the relationship between the levels of speech intensity that were produced for each level of altered intensity feedback in the different speech tasks. A two-way ANOVA (group by speech task) was used to examine the average AIF slopes in the different speech tasks for the PD and control groups.

To examine the effect of the magnitude production task on speech intensity and the response to AIF, a two-way ANOVA with magnitude production conditions (habitual loudness, 2x loudness, 4x loudness, maximum loudness) and group (PD vs. healthy controls) was first used to examine the effects of MP conditions on speech intensity in the PD and control groups. A three-way repeated measures ANOVA was then used to analyze the effect of MP task conditions on the AIF levels in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and magnitude production conditions (habitual loudness, 2x loudness, 4x loudness, maximum loudness) and AIF level (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB), as within-subjects factors. Any significant interactions lead to post-hoc comparisons.

To examine the effect of the imitation task on speech intensity and the response to AIF, a two-way ANOVA with imitation task levels (50dB, 60dB, 70dB, 80dB) and group (PD vs. healthy controls) was first used to examine the effects of imitation levels on speech intensity in the PD and control groups. A three-way repeated measures ANOVA was then used to analyze the effect of imitation task levels on the AIF conditions in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and imitation task levels (50dB, 60dB, 70dB, 80dB) and AIF level (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB), as within-subjects factors. Any significant interactions lead to post-hoc comparisons.

To examine the effect of the instruction to ignore auditory feedback task on speech intensity and the response to AIF, a two-way ANOVA with instruction conditions (no instruction, with instruction) and group (PD vs. healthy controls) was first used to examine the effects of instruction conditions on speech intensity in the PD and control groups. A three-way repeated measures ANOVA was then used to analyze the effect of instruction condition on the AIF levels in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and instruction condition (no instruction, with instruction) and AIF level (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB), as within-subjects factors. Any significant interactions lead to post-hoc comparisons.

2.4.3 Statistical Analysis for 65dB Background Noise

The speech intensity responses to 65dB of background noise in the PD and control groups were analyzed using a two-way ANOVA with group (PD vs. healthy controls) as the between-subjects factor, and background noise (no noise, 65dB background noise) as the within-subjects factor. This analysis was completed separately for the different speech tasks (conversation at a near distance, conversation at a far distance, vowel, reading), the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness), the imitation task (50dB, 60dB, 70dB, 80dB), and the instruction conditions (no instruction, with instruction to ignore auditory feedback). Any significant interactions lead to post-hoc comparisons.

A three-way repeated measures ANOVA was used to analyze the effect of background noise on the speech tasks (conversation, vowel, reading) in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and background noise (no noise, 65dB background noise) and speech tasks (conversation, vowel, reading) as within-subjects factors. This analysis was also completed for the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness), the imitation task (50dB, 60dB, 70dB, 80dB) and the instruction conditions (no instruction, with instruction to ignore auditory feedback). Any significant interactions lead to post-hoc comparisons.

A three-way repeated measures ANOVA was used to analyze the effect of background noise on the AIF conditions in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and background noise (no noise, 65dB background noise) and AIF level (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB), as within-subjects factors. Any significant interactions lead to post-hoc comparisons.

Linear regression analysis was completed in order to examine the relationship between the levels of speech intensity that were produced for each level of AIF in the context of background noise. A two-way ANOVA (group by noise condition) was used to examine the average AIF slopes in the different noise conditions for the PD and control groups.

2.4.4 Statistical Analysis for Complete Masking Noise

Responses to complete masking noise were subjected to a two-way with group (PD vs. healthy controls) as the between-subjects factor and masking noise condition (no noise, 100dB complete masking noise) as the within-subjects factor. This analysis was completed separately for the speech tasks (conversation at a near distance, conversation at a far distance, vowel, reading), the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness), and the imitation task (50dB, 60dB, 70dB, 80dB).

A three-way repeated measures ANOVA was used to analyze the effect of masking noise on the speech tasks (conversation, vowel, reading) in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and with masking noise condition (no noise, 100dB complete masking noise) and speech tasks (conversation at a near distance, conversation at a far distance, vowel prolongation, reading) as within-subjects factors. This analysis was also completed for the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness), and the imitation task (50dB, 60dB, 70dB, 80dB). Any significant interactions lead to post-hoc comparisons.

2.4.5 Statistical Analysis for Self-Loudness Perception Ratings

Participant loudness perception ratings on the VAS in the MP task will be measured and subjected to a two-way ANOVA involving the MP conditions (habitual loudness, 2x louder, 4x louder, maximum loudness) and group factors (PD vs. controls). A two-way ANOVA involving group (PD vs. controls) and AIF levels (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB) was used to examine the self-loudness ratings across the AIF levels in the MP task. A three-way repeated measures ANOVA was used to analyze the ratings in the MP task across the AIF levels with group (PD vs. controls) as the between subjects factor, and with AIF conditions (-15dB, -10dB, -5dB, 0dB, +5dB, +10dB, +15dB), and MP levels (habitual loudness, 2x louder, 4x louder, maximum loudness) as within-subjects factors.

The self-loudness perception ratings in 65dB of background noise in the PD and control groups were analyzed using a two-way ANOVA with group (PD vs. healthy controls) as the between-subjects factor, and background noise (no noise, 65dB background noise) as the within-subjects factor. This analysis was completed separately for the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness), and the instruction to ignore auditory feedback (no instruction, with instruction to ignore auditory feedback). Any significant interactions lead to post-hoc comparisons.

A three-way repeated measures ANOVA was used to analyze the effect of background noise on the loudness ratings in the MP task in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and background noise (no noise, 65dB background noise) and MP tasks ((habitual loudness, 2x louder, 4x louder, maximum loudness) as within-subjects factors. This analysis was also completed for the instruction to ignore auditory feedback (no instruction, with instruction to ignore auditory feedback). Any significant interactions lead to post-hoc comparisons.

Self-loudness perception ratings during the MP task in complete masking noise were subjected to a two-way with group (PD vs. healthy controls) as the between-subjects factor and masking noise condition (no noise, 100dB complete masking noise) as the within-subjects factor.

A three-way repeated measures ANOVA was used to analyze the loudness ratings during the MP task in complete masking noise in the two groups, with group (PD vs. healthy controls) as the between-subjects factor, and with masking noise condition (no noise, 100dB complete masking noise) and MP tasks (habitual loudness, 2x louder, 4x louder, maximum loudness) as within-subjects factors. Any significant interactions lead to post-hoc comparisons.

Chapter 3: Results

3.1 Altered Intensity Feedback (AIF)

3.1.1 Effect of AIF on speech intensity in PD and HC groups (Objective 1)

The primary objective of the present study was to examine the effect of altered intensity feedback (AIF) on speech intensity in PD. This objective was addressed by comparing the effects of seven AIF conditions on speech intensity in PD and HC groups. The statistical method used to address this objective was the two-way repeated measures ANOVA. The statistical result that best addressed objective 1 was the two-way interaction involving group by AIF condition interaction.

The marginal means related to the 7 feedback conditions for both the PD and control groups are shown in Figure 2 and the related descriptive statistics are shown in Table 1. The results of the two-way (group by AIF feedback condition) ANOVA indicated that there was no significant main effect of group (F (1,46) = 0.327, p = 0.570) with PD participants having a similar marginal mean (M = 68.204; SD = 2.98 to that of the control participants (M = 68.639; SD = 2.21). In contrast, there was a significant main effect of altered intensity feedback condition on speech intensity (F (6,276) =197.48, p = 0.000). A post-hoc analysis was used to examine the pairwise comparisons related to the 7 feedback conditions. The results of these pairwise post-hoc comparisons are shown in Table 2. In general, a significant difference in speech intensity was found for each of the pairwise comparisons and this is reflected in the general pattern involving a gradual increase in response speech intensity as the intensity of the altered feedback was gradually reduced from +15 to -15dB (Figure 2).

It is important to note that the main effect of feedback condition needs to be qualified because of the finding of a significant group by feedback condition interaction (F (6,276) = 42.55, p = 0.000) for speech intensity. This significant interaction is illustrated in Figure 2, which shows that the previously described trend involving an increase in speech intensity as the feedback intensity decreases is different for the PD participants relative to the control participants. In particular, across the feedback conditions, the control participants showed greater response intensity to the feedback conditions than the PDs. This is also reflected in Figure 2 by the steeper negative slope in the intensity versus feedback condition plot for the controls relative to the PD participants.

In order to examine the group by feedback condition interaction in more detail, difference values were calculated by subtracting the response speech intensity produced during the 0dB altered feedback condition from each of the other 6 altered intensity feedback conditions (-15dB, -10dB, -5dB, +5dB, +10dB, +15dB). These difference results are shown in Figure 3. Each of these 6 zero-referenced difference scores were obtained for the PD and control participants and submitted to an interaction post-hoc analysis. This interaction post-hoc analysis revealed a significant group difference for 5 of the 6 pairs (exception is the +5 - 0dB difference) of the condition difference comparisons ($p < .05$). The descriptive statistics and the results of this interaction-related post-hoc analysis are presented in Table 3. The results of this post-hoc analysis revealed that the size of the compensation response (i.e. zero referenced difference score), both in the negative and positive directions was consistently lower for the PD participants relative to the controls.

An additional interaction-related post-hoc analysis was performed to compare the absolute size of the compensation response for the negative feedback conditions to that of the positive feedback conditions and to compare this negative versus positive feedback difference across the PD and control groups. The results of this interaction related post-hoc analysis is presented in Table 4. The results indicate the absolute size of the response intensity is smaller for the -15dB feedback condition than the +15dB feedback condition for both the PD group and the control group (p = .001; p = .029 respectively). Interestingly, although the following comparisons did not reach statistical significance, for the -10dB vs. +10dB comparison, the absolute size of the response intensity was larger for the negative feedback condition compared to the positive feedback condition in the control group but smaller for the negative feedback condition in the PD group.

Figure 2. ***Marginal means for PD and HC groups and the 7 AIF conditions.***

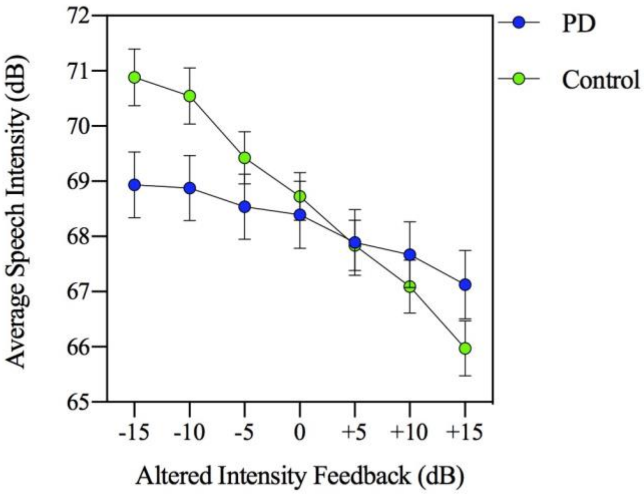


Table 2. ***Marginal means and standard deviations related to the 7 AIF conditions obtained for the PD (n= 25) and HC (n=23) groups.***

AIF Conditions	PD			HC	
	Mean	*SD*		Mean	*SD*
-15 dB	68.93	2.99		70.88	2.48
-10 dB	68.88	2.97		70.54	2.45
-5 dB	68.54	2.96		69.43	2.25
0 dB	68.39	3.06		68.72	2.09
+5 dB	67.89	2.98		67.84	2.18
+10 dB	67.67	3.00		67.09	2.29
+15 dB	67.13	3.10		65.97	2.37

Table 3. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 7 AIF conditions (-15dB), (-10dB), (-5dB), (0dB), (+5dB), (+10dB), and (+15dB).***

Feedback Conditions			Pairwise comparisons and p values						
	Mean	*SD*	-15	-10	-5	0	+5	+10	+15
-15 dB	69.91	2.76							
-10 dB	69.71	2.73	.291						
-5 dB	68.98	2.65	**<.001***	**<.001***					
0 dB	68.56	2.64	**<.001***	**<.001***	**<.001***				
+5 dB	67.87	2.63	**<.001***	**<.001***	**<.001***	**<.001***			
+10 dB	67.38	2.69	**<.001***	**<.001***	**<.001***	**<.001***	**<.001***		
+15 dB	66.55	2.78	**<.001***	**<.001***	**<.001***	**<.001***	**<.001***	**<.001***	

** = significant at* $p < 0.05$

Figure 3. ***Difference values for PD and HC groups and 6 AIF conditions.***

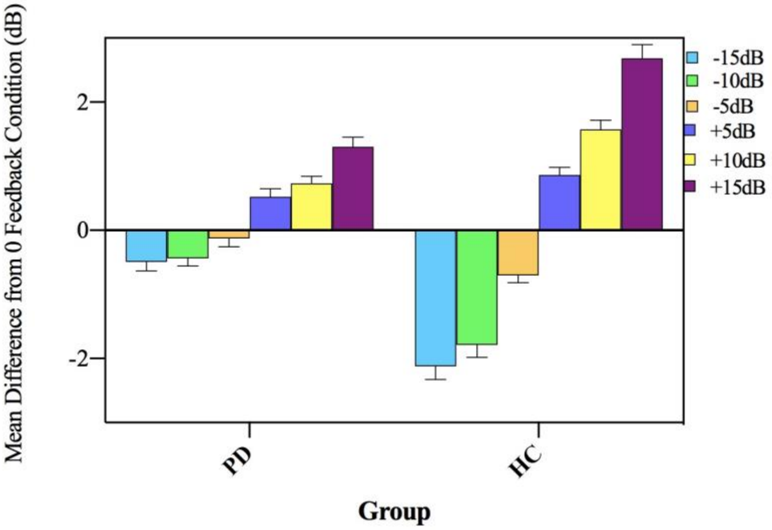


Table 4. ***Post-hoc comparisons related to difference scores (referenced to the 0 feedback condition) for the 6 difference conditions (-15 – 0), (-10 – 0), (-5 – 0), (5 – 0), (10 – 0), and (15-0) obtained for the PD (n=26) and HC (n=24) groups.***

Difference Conditions	PD			HC			PD – HC difference score t-test			*p value*
	Mean	*SD*		Mean	*SD*		Mean difference	Standard error difference	t-value	
-15 – 0	-.49	.73		-2.12	1.02		1.64	.25	t(48) = 6.58	**<.001***
-10 – 0	-.44	.59		-1.79	.94		1.35	.22	t(48) = 6.11	**<.001***
-5 – 0	-.13	.66		-.70	.58		.57	.18	t(48) = 3.25	**.002***
5 – 0	.52	.64		.86	.61		-.34	.18	t(48) = -1.91	.063
10 – 0	.73	.58		1.57	.72		-.84	.18	t(48) = -4.59	**<.001***
15 – 0	1.30	.79		2.68	1.06		-1.38	.26	t(48) = -5.25	**<.001***

** = significant at $p < 0.05$*

Table 5. ***Post-hoc comparisons related to difference scores (referenced to the 0 feedback condition) for the 3 difference conditions related to the negative versus positive feedback conditions (-15 vs +15), (-10 vs +10), and (-5 vs +5) obtained for the PD (n=26) and HC (n=24) groups.***

Difference Conditions	PD			HC			PD – HC difference score		*p value*
	Mean	*SD*		Mean	*SD*		Mean	SD	
-15 vs +15	1.78	1.05		4.80	1.71		.61	1.03	**.002***
-10 vs +10	1.18	.75		3.36	1.33		.003	.81	1.000
-5 vs +5	.65	.61		1.56	.77		.112	.76	1.000

* = significant at $p < 0.05$

3.1.2 AIF slope analysis of PD and HC groups

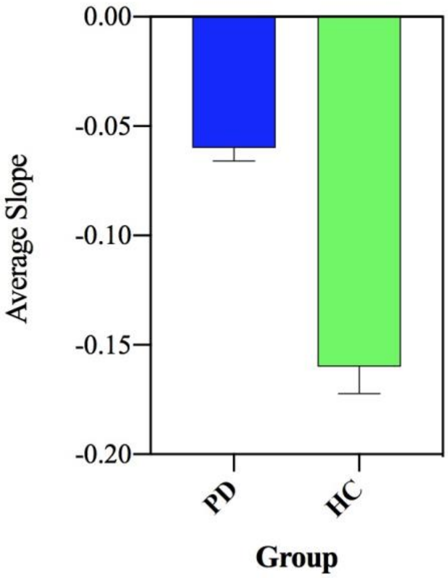
To examine the effect of AIF conditions in more detail, a linear regression analysis was performed on each participant’s data using the speech intensity response values and the corresponding values relating to each of the AIF conditions (-15 to +15dB). From each of these individual participant regression analyses an individual slope value was obtained. Thus, the slope of the AIF response function was determined for each participant and these individual slope values were used to compare the average AIF slope for the PD and control groups using an independent t-test. The results of the t-test indicated a significant difference between groups (t (48) = 8.174, p = 0.000), such that the PD group had a significantly reduced negative slope (M= -0.06; SD= 0.03) compared to the steeper negative slope of the control group (M= -0.16; SD= 0.06). The descriptive statistics related to this analysis are provided in Table 5 and depicted in Figure 4.

Table 6. ***Descriptive statistics related to the slope values in the PD and HC groups.***

PD			HC			PD-HC t-test			*p value*
Mean	*SD*		Mean	*SD*		Mean difference	SE difference	t value	
-0.06	0.03		-0.16	0.06		0.10	0.01	t(48) = 8.17	**<.001***

** = significant at p < 0.05*

Figure 4. ***Average slope values in the PD and HC groups.***



3.2 Speech Tasks

<u>3.2.1 Effect of speech tasks on speech intensity and the response to AIF in PD and HC groups (Objective 2)</u>

Objective 2 had a dual purpose. The first part was to examine the effects of different speech tasks on speech intensity in PD and HC groups. The second part was to examine the effects of speech tasks on the response to the AIF conditions in the PD and

HC groups. In order to address part 1, a two-way ANOVA involving speech task and group factors was used.

The results of the two-way (group by speech task) ANOVA indicated that there was not a significant main effect of group (*F* (1,46) = .327, *p* = 0.570). The results of the two-way ANOVA indicated that there was a significant main effect of speech task (*F* (3,138) = 82.08, *p* = 0.000). The descriptive statistics related to the speech intensity obtained for the PD and HC groups during each of the speech tasks are shown in Table 6. The post hoc analysis of simple main effects related to the 4 speech tasks (conversation at a near distance, conversation at a far distance, vowel prolongation, and reading task) are shown in Table 7. In general, post hoc analysis of simple main effects for speech tasks revealed that speech intensity was increased in conversation at a far distance compared to conversation at a near distance and reading sentences. In addition, the sentence reading task had lower speech intensity than all other tasks. The group by speech task interaction was not significant (F (3,138) = 1.335, p = .265).

Table 7. ***Descriptive statistics for the 4 speech tasks in the PD and HC groups.***

Speech Task	PD		HC	
	Mean	*SD*	Mean	*SD*
Conversation (near)	67.90	3.31	68.53	2.85
Conversation (far)	70.07	3.5	71.30	2.81
Vowel	69.78	3.27	69.95	2.06
Reading (habitual)	65.07	3.91	64.77	2.82

Table 8. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 4 speech tasks.***

Speech Task			Pairwise comparisons and p values			
	Mean	*SD*	Conversation (near)	Conversation (far)	Vowel	Reading (habitual)
Conversation (near)	68.22	3.07				
Conversation (far)	70.68	3.16	**<.001***			
Vowel	69.87	2.73	**<.001***	.414		
Reading (habitual)	64.92	3.40	**<.001***	**<.001***	**<.001***	

** = significant at $p < 0.05$*

The above results indicate that there was an effect of the speech tasks on speech intensity. Given this speech task effect, an important consideration is to determine if this speech task effect had a modulating effect on the AIF conditions in the PD and HC groups. This consideration is the focus of the second part of Objective 2. In order to examine this potential modulating effect, a three-way (group by AIF feedback condition by speech task) repeated measures ANOVA for the dependent measure of speech intensity was performed. The results of the three-way ANOVA indicated that there was a significant three-way interaction involving group, altered intensity feedback condition and speech task (F (18,828) =10.631, p = 0.000). A significant three-way interaction indicates that an underlying two-way interaction differs as a function of a third factor or independent variable. In the present context, the three-way interaction indicated that the two-way interaction involving the AIF condition by group interaction differed across the third factor related to speech tasks.

In order to interpret this three-way interaction, a separate plot of the two-way AIF by group interaction was created for each of the four speech conditions. These four plots are shown in figures 5, 6, 7 and 8. The descriptive statistics related to the data in these figures is presented in Table 8. Visual inspection of these four figures indicates how the two-way interaction involving AIF by group differed across the speech tasks. As previously described, the two-way AIF by group interaction was characterized by the control group having a steeper negative slope than the PD group across the AIF conditions (-15dB to +15dB). When the four speech tasks are examined separately it is observed that the group difference in the slopes is not the same across the four speech tasks. In particular, the group difference in the slopes was more pronounced during the conversation tasks than during the reading and vowel tasks. Thus, for the conversation tasks, the control group had a much steeper negative slope than the PD group but, for the reading and vowel tasks, the control group had a negative slope that was similar to that of the PD group. In general, these results indicate that the PD participants had a different response to the AIF conditions than the controls and that this abnormal response to AIF was modulated by the speech tasks and was most apparent during the conversation tasks.

Table 9. ***Descriptive statistics for the 4 speech tasks and AIF levels in the PD and HC groups.***

Speech Task	AIF Level	PD			HC	
Conversation (near distance)		Mean	*SD*		Mean	*SD*
	-15dB	68.18	3.29		71.37	3.40
	-10dB	68.25	3.35		71.04	3.56
	-5dB	68.25	3.47		69.25	2.84
	0dB	67.89	3.41		68.48	2.56
	+5dB	67.68	3.46		67.49	3.07
	+10dB	67.81	3.47		66.91	2.85
	+15dB	67.26	3.41		65.18	2.93

Conversation (far distance)	-15dB	70.60	3.78		74.10	3.12
	-10dB	70.44	3.92		73.34	2.85
	-5dB	70.45	3.56		72.28	2.82
	0dB	70.29	3.47		71.43	2.97
	+5dB	70.09	3.46		70.52	2.82
	+10dB	69.31	3.48		69.34	2.96
	+15dB	69.31	3.53		68.08	3.05
Vowel Prolongation	-15dB	71.32	3.07		72.15	2.36
	-10dB	71.04	2.99		71.87	2.33
	-5dB	70.31	3.16		71.08	2.35
	0dB	69.88	3.53		69.84	1.93
	+5dB	69.32	3.53		69.12	2.13
	+10dB	68.51	3.39		68.05	2.02
	+15dB	68.07	3.95		67.57	2.52
Reading (habitual)	-15dB	65.63	3.96		65.91	3.07
	-10dB	65.78	3.79		65.92	3.09
	-5dB	65.13	4.07		65.09	3.07
	0dB	65.50	3.96		65.15	2.80
	+5dB	64.49	4.08		64.22	2.65
	+10dB	65.06	4.13		64.06	3.04
	+15dB	63.87	3.79		63.05	2.71

Figure 5. ***Marginal Means for the PD and HC groups across 7 AIF conditions in the conversation at a near distance speech task.***

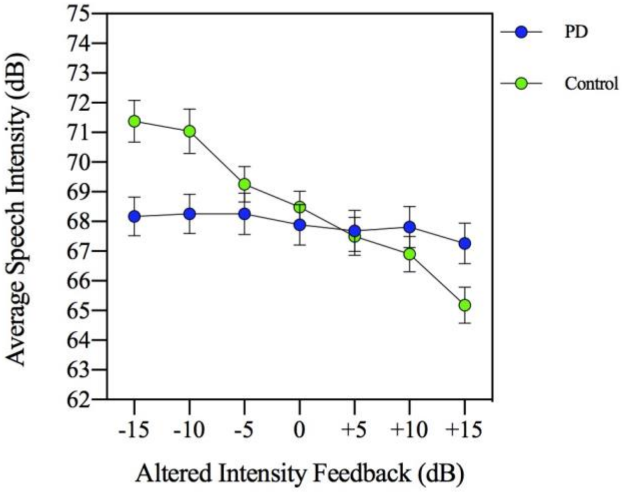


Figure 6. ***Marginal Means for the PD and HC groups across 7 AIF conditions in the conversation at a far distance speech task.***

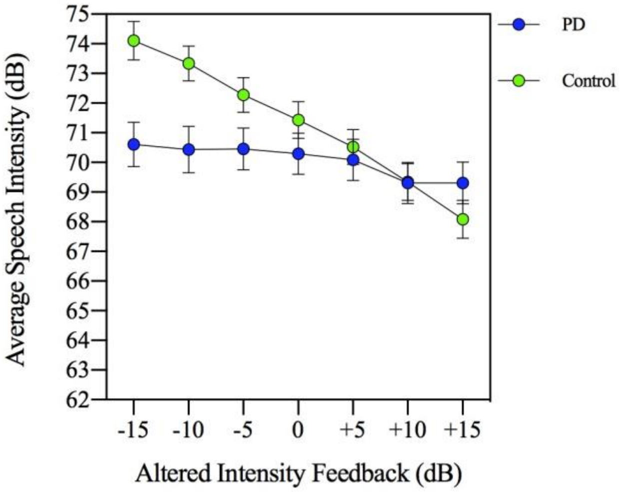


Figure 7. ***Marginal Means for the PD and HC groups across 7 AIF conditions in the vowel prolongation speech task.***

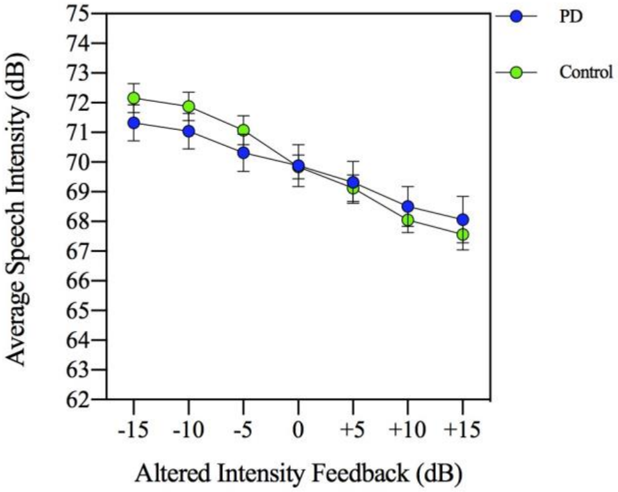
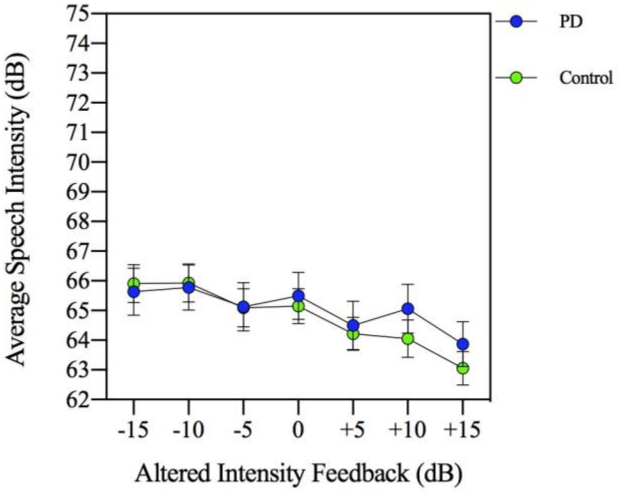


Figure 8. ***Marginal Means for the PD and HC groups across 7 AIF conditions in the sentence reading (habitual speech intensity) speech task.***



3.2.2 AIF slope analysis of speech tasks

To examine the effect of speech tasks on the AIF conditions in more detail, the slope of the AIF function was determined for each participant and the average slope for each group was examined across the 4 speech tasks. For this slope analysis, the average slope was examined using a two-way (group by speech task) ANOVA. The results indicated a main effect of group (*F* (1,43) = 60.59, *p* = 0.000), such that the PD group had a significantly lower (flatter) slope (M= -0.061; SD= 0.04) compared to the steeper slope of the control group (M= -0.167; SD= 0.05). In addition, results of the two-way ANOVA revealed a significant main effect of speech task (F (3, 129)= 17.434, p = 0.000). The descriptive statistics related to the slope of each speech task (conversation at a near distance, conversation at a far distance, vowel prolongation, reading at habitual loudness) for both the PD and control groups are shown in Table 9. These results suggest that overall; the participants produced a flatter slope of the AIF response function in the reading task compared to all other speech tasks. The results for the two-way ANOVA also produced a significant group by speech task interaction F (3, 129) = 26.959, p = .000. The descriptive statistics related to the slope values of the 4 speech tasks in the PD and control groups are provided in Table 10 and depicted in Figure 9.

Table 10. ***Post-hoc results related to pairwise comparisons involving the marginal slope means for the 4 speech tasks.***

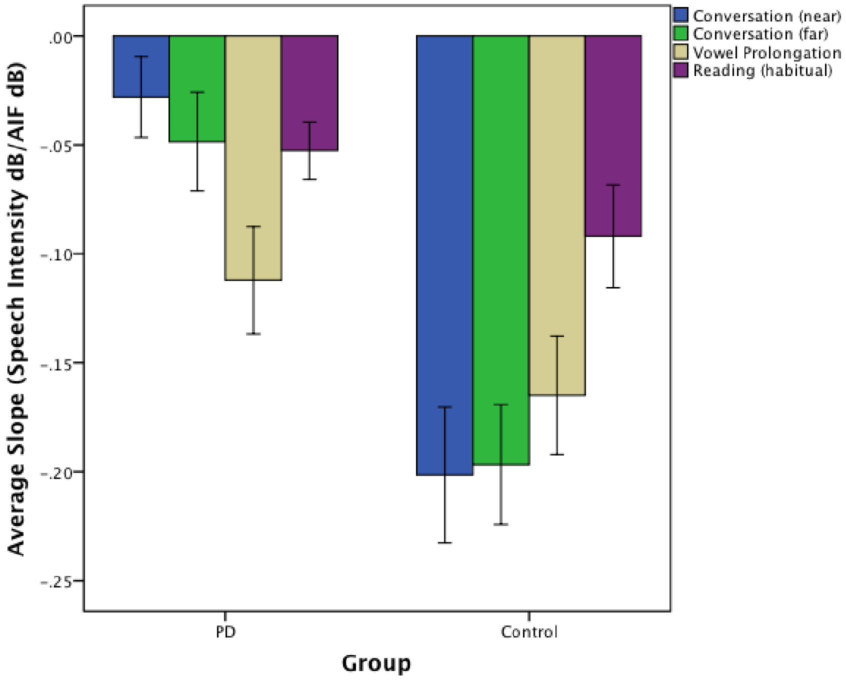
Speech Task			Pairwise comparisons and p values			
	Mean	*SD*	Conversation (near)	Conversation (far)	Vowel	Reading (habitual)
Conversation (near)	-.118	.07				
Conversation (far)	-.120	.05	1.000			
Vowel	-.142	.07	.243	.434		
Reading (habitual)	-.075	.05	**<.001***	**<.001***	**<.001***	

** = significant at* $p < 0.05$

Table 11. ***Descriptive statistics for the slope values of the 4 speech tasks in the PD and HC groups.***

Speech Task	PD		HC	
	Mean	*SD*	Mean	*SD*
Conversation (near)	-.032	.05	-.205	.08
Conversation (far)	-.042	.06	-.198	.06
Vowel	-.116	.07	-.167	.07
Reading (habitual)	-.054	.03	-.097	.06

Figure 9. Marginal slope mean for the 4 speech tasks by the PD and HC group.



Error bars: +/- 2 SE

To examine this interaction in more detail, post hoc analysis was performed. This post-hoc analysis involved comparisons between the PD and control groups for each of the pairwise difference scores related to the 4 speech tasks. Results of the post-hoc analysis are provided in Table 11. This post-hoc analysis revealed that the group differences in slope values for the speech tasks was most apparent in the conversation compared to the other speech tasks (vowel and reading), and that neither the difference in conversation at near vs. far interlocutor distances nor the difference between vowel and reading speech tasks differed significantly between groups. Thus, the slope analysis is consistent with the AIF level analysis and further confirms that the AIF function is steeper in controls and that this group difference is most apparent in the conversational speech tasks rather than the reading and vowel tasks.

Table 12. ***Interaction post-hocs involving a comparison of the two groups (PD vs. HC) for each of the 6 pairwise difference scores related to the 4 speech tasks (convN – convF), (conN – read), (convN – vowel), (convF – read), (convF – vowel), and (read – vowel).***

Difference Conditions	PD			HC			PD – HC difference score t-test			*p value*
	Mean	*SD*		Mean	*SD*		Mean difference	Standard error difference	t-value	
ConN - ConF	-.02	.06		.00	.07		-.03	.02	t(48) = -1.34	.175
ConN – Vowel	-.08	.07		.04	.07		-.12	.02	t(48) = -6.05	**<.001***
ConN – Read	-.02	.05		.11	.05		-.13	.01	t(48) = -9.74	**<.001***
ConF – Vowel	-.06	.09		.03	.07		-.10	.02	t(48) = -4.01	**<.001***
ConF – Read	-.00	.06		.10	.07		-.11	.02	t(48) = -6.07	**<.001***
Read – Vowel	.06	.06		.07	.05		-.01	.01	t(48) = -.93	.358

* = *significant at* $p < 0.05$

3.3 Background Noise

3.3.1 The effects of background noise conditions on speech intensity and the response to AIF in PD and HC groups (Objective 3)

Objective 3 was focused on two parts. The first part was to examine the effects of different background noise conditions on speech intensity in PD and HC groups. The second part was to examine the effects of noise conditions on the response to the AIF conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving noise conditions and group factors was used.

The descriptive statistics related to the noise conditions (no noise and 65dB background noise) for both the PD and control groups are shown in Table 12. The results of the two-way (group by background noise) ANOVA indicated that there was a significant main effect of BGN (*F* (1,46) = 25.725, *p* = 0.000). Post hoc analysis of simple main effects revealed that the noise condition (M = 69.28; SD = 2.70) was associated with higher speech intensity relative to the no noise condition (M = 67.56; SD = 3.06) (p = .000). The group by noise condition interaction was not significant (F (1,46) = 2.185, p = .146).

Table 13. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions obtained for the PD (n= 25) and HC (n=23) groups.***

Background Noise	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	67.60	3.43		67.53	2.58
65 dB noise	68.81	3.05		69.75	2.26

The above results indicate that there was an effect of the noise conditions on speech intensity. Given this noise condition effect, an important consideration was to determine if the noise conditions had a modulating effect on the AIF conditions in the PD and HC groups. This consideration is the focus of the second part of Objective 2. In order to examine this potential modulating effect, a three-way (group by AIF feedback condition by noise condition) repeated measures ANOVA for the dependent measure of speech intensity was performed.

The results of the three-way ANOVA indicated that there was a significant three-way interaction involving altered intensity feedback condition, background noise and group on speech intensity (*F* (6,276) =4.202, *p* = 0.000). This significant three-way interaction indicates that an underlying two-way interaction differs as a function of a third factor or independent variable. In the present context, the three-way interaction indicates that the two-way interaction involving the AIF condition by group interaction differs across the third factor related to the noise conditions.

In order to interpret this three-way interaction, a separate plot of the two-way AIF by group interaction was created for both of the noise conditions. These two plots are shown in Figures 10 and 11. The descriptive statistics related to the data in these figures is presented in Table 13. Visual inspection of these two figures indicates how the two-way interactions involving AIF by group differ across the noise conditions. As previously described, the two-way AIF by group interaction is characterized by the control group having a steeper negative slope than the PD group across the AIF conditions (-15dB to +15dB). However, when the two noise conditions are examined separately it is observed that the group difference in the slopes is not the same across the noise conditions. In particular, the group difference in the slopes is more pronounced during the 65dB noise condition than during the no noise condition. Thus, for the noise condition, the control group has a much steeper negative slope than the PD group but, for no noise condition, the control group has a negative slope that is similar to that of the PD group. In general, these interaction results indicate that the PD participants had a different response to the AIF conditions than the controls and that this abnormal response to AIF was most apparent during the 65dB noise condition.

Figure 10. ***Marginal Means for the PD and HC groups for the no noise condition across 7 AIF conditions.***

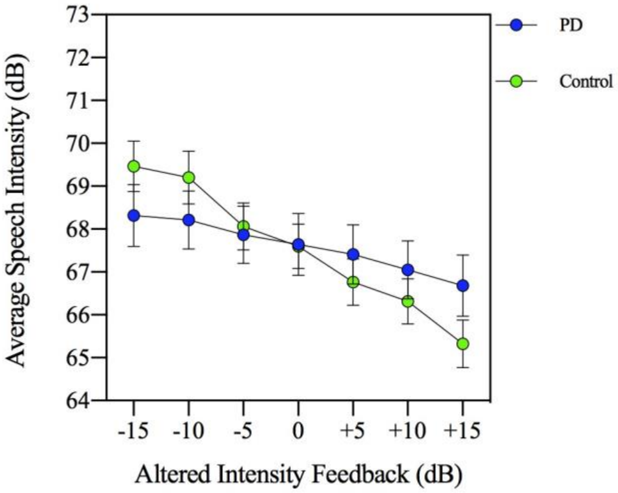


Figure 11. ***Marginal Means for the PD and HC groups across 7 AIF conditions for the 65dB background noise condition.***

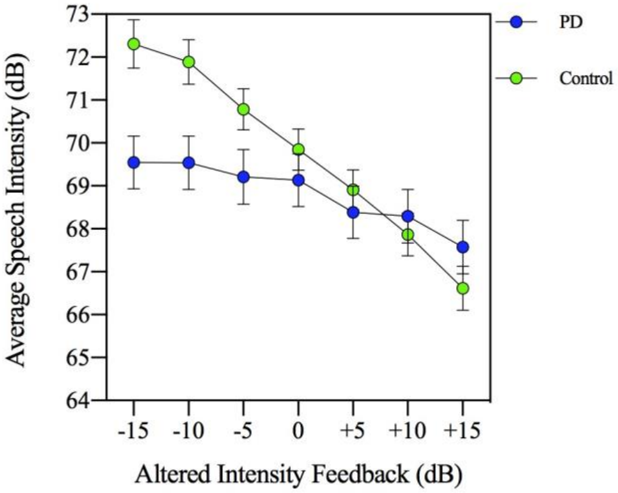


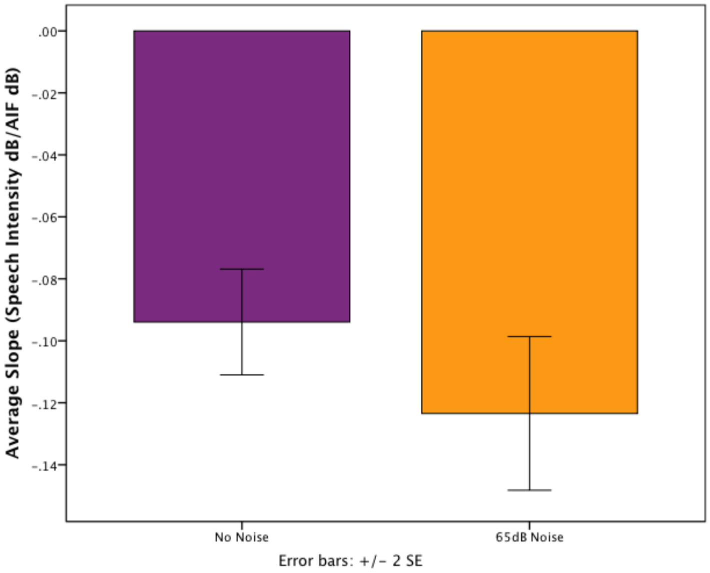
Table 14. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions and AIF conditions obtained for the PD (n= 25) and HC (n=23) groups.***

Background Noise Condition	AIF Level	PD			HC	
No Noise		Mean	*SD*		Mean	*SD*
	-15dB	68.32	3.59		69.46	2.80
	-10dB	68.21	3.38		69.20	2.97
	-5dB	67.87	3.35		68.06	2.65
	0dB	67.65	3.62		67.60	2.48
	+5dB	67.41	3.48		66.76	2.59
	+10dB	67.05	3.37		66.31	2.55
	+15dB	66.68	3.59		65.32	2.65
65dB Noise	-15dB	69.55	3.08		72.31	2.69
	-10dB	69.54	3.13		71.88	2.50
	-5dB	69.21	3.20		70.79	2.30
	0dB	69.14	3.09		69.85	2.31
	+5dB	68.38	3.03		68.91	2.24
	+10dB	68.29	3.11		67.87	2.38
	+15dB	67.58	3.13		66.62	2.46

<u>*3.3.2 AIF slope analysis of noise conditions*</u>

To examine the effect of the noise conditions on the AIF conditions in more detail, the slope of the AIF function was determined for each participant and the average slope for each group was examined across the two noise conditions. For this slope analysis, the average slope was examined using a two-way (group by noise condition) ANOVA. The results indicated a main effect of group (F (1,43) = 60.59, p = 0.000), such that the PD group had a significantly less steep negative slope (M= -0.061; SD= 0.04) compared to the steeper negative slope in the control group (M= -0.167; SD= 0.05). In addition, results of the two-way ANOVA revealed a significant main effect of background noise (F (1, 43) = 11.717, p = 0.001), such that a significantly less steep slope was produced by both groups in no noise condition (M= -0.10; SD= 0.05) compared to the steeper slope produced in the 65dB noise condition (M= -0.13; SD= 0.06). These findings are also depicted in Figure 12.

Figure 12. ***Marginal means related to the AIF slope for the 2 background noise conditions. The dB/dB unit of the slope variable corresponds to the dB reduction in speech intensity per dB increase in AIF.***



Results of the ANOVA revealed an interaction effect of background noise and group (F (1, 43) = 5.354, p= .026). Figure 13 presents these findings and these are also reflected in the post hoc analysis of the interaction (Table 14), which revealed that the PD group had slope values that were similar across the noise conditions while the control group showed a difference in slope values across the noise conditions. This slope analysis indicates that the control participants produce a steeper AIF function compared to the PD participants and that this group difference in the AIF slope becomes more apparent in the context of background noise.

Figure 13. ***Marginal slope means for the 2 noise conditions in the PD and HC groups. The dB/dB unit of the slope variable corresponds to the dB reduction in speech intensity per dB increase in AIF.***

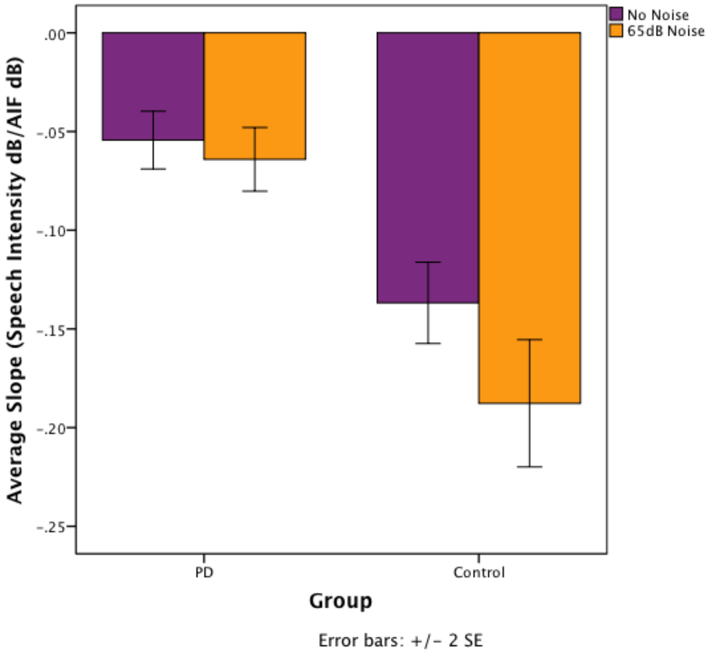


Table 15. ***Interaction post-hocs involving a comparison of the two groups (PD vs. HC) for each of the difference scores related to the two noise conditions (no noise – 65dB noise).***

Difference Conditions	PD			HC			PD – HC difference score t-test			*p* *value*
	Mean	*SD*		Mean	*SD*		Mean difference	Standard error difference	t-value	
No Noise – Noise	-.01	.05		-.05	.07		.04	.02	t(48)= 2.44	**.019***

** = significant at* $p < 0.05$

3.4 Magnitude Production (MP) Task

3.4.1 Effect of MP task on speech intensity and the response to AIF in PD and HC groups (Objective 4)

Objective 4 was focused on two parts. The first part was to examine the effects of a Magnitude Production (MP) task on speech intensity in PD and HC groups. The second part was to examine the effects of the MP task levels on the response to the AIF conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the MP levels (habitual loudness, 2 times louder, 4 times louder, maximum loudness) and group factors was used.

The results of the two-way (group by MP task levels) ANOVA indicated that there was a significant main effect of the MP task (F (3,144) = 330.395, p = 0.000). The descriptive statistics related to the MP task levels (habitual loudness, 2 times louder, 4 times louder, maximum loudness) for all participants are shown in Table 15 and depicted in Figure 14. As the table and figure suggest, the speech intensity produced by participants increased with each successive magnitude production level (p=.000). Both the main effect of group (F(1,46)= .591, p= .446) and the group by MP task interaction (F (3,144) = 2.400, p = .070) were not significant. The two-way interaction involving group by AIF conditions was significant (F(6,288)=9.207, p=.000). As depicted in Figure 15, this interaction confirms that the PD and HC groups continued to show different responses (reduced slope by the PD group) to the AIF conditions even in the context of the 4 magnitude production tasks.

Table 16. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 4 MP levels.***

Magnitude Production Level			Pairwise comparisons and p values			
	Mean	*SD*	Habitual	2x louder	4x louder	Maximum
Habitual	64.86	3.38				
2 x louder	69.06	3.91	**<.001***			
4 x louder	72.28	4.37	**<.001***	**<.001***		
Maximum	75.57	4.34	**<.001***	**<.001***	**<.001***	

** = significant at p < 0.05*

Figure 14. ***Marginal means for PD and HC groups and the 4 MP task levels.***

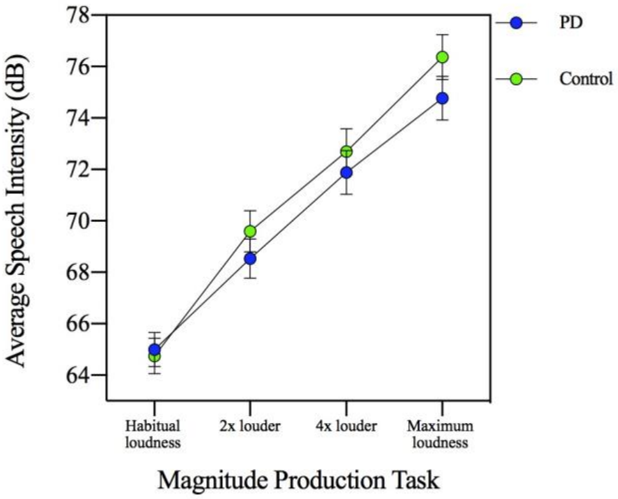
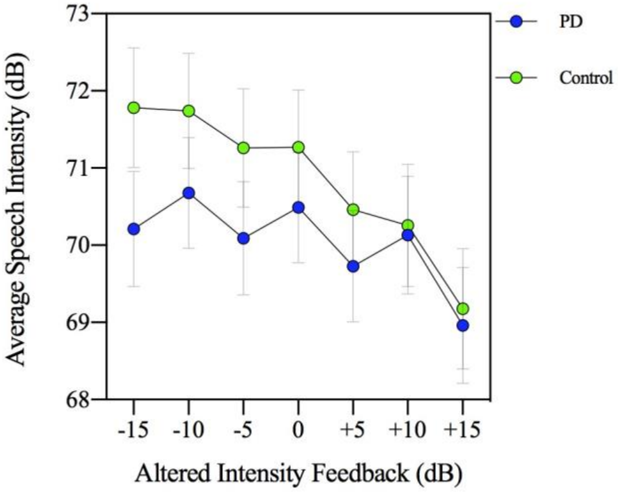


Figure 15. ***Marginal means for PD and HC groups and the 7 AIF conditions in the MP task.***



Given there was an effect of the MP task on speech intensity, the second part of Objective 4 aimed to examine the potential modulating effect of this MP task on the AIF conditions in the two groups. A three-way (group by AIF feedback condition by MP level) repeated measures ANOVA for the dependent measure of speech intensity was performed. The three-way ANOVA results indicate the group by AIF task by MP task interaction only approached significance (F (18,864) = 1.495, p= .084), suggesting that the MP levels did not have a modulating effect on the AIF conditions in the PD and HC groups. This three-way interaction is depicted in Figures 16, 17, 18, and 19 and it appears this interaction approached significance as a result of the speech intensity produced in the maximum loudness MP condition. As depicted in Figure 18, the PD group produced a flatter slope across the AIF levels compared to the control group in this condition, perhaps due to a further reduction of the AIF effects while producing maximum loudness.

Figure 16. ***Marginal means for the PD and HC groups across 7 AIF conditions in the reading at habitual loudness MP task.***

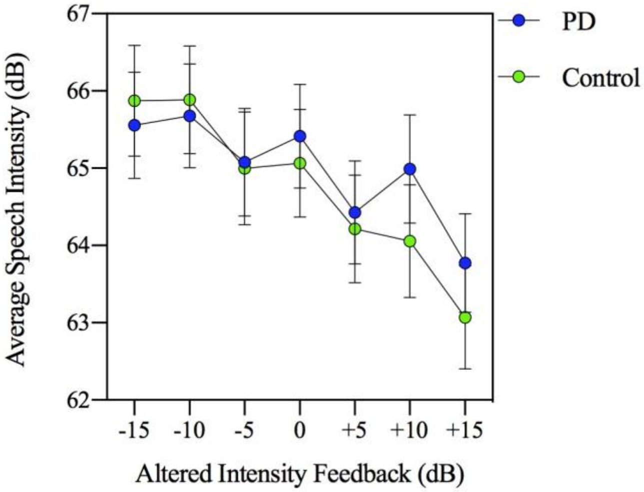


Figure 17. ***Marginal means for the PD and HC groups across 7 AIF conditions in the reading at 2x louder MP task.***

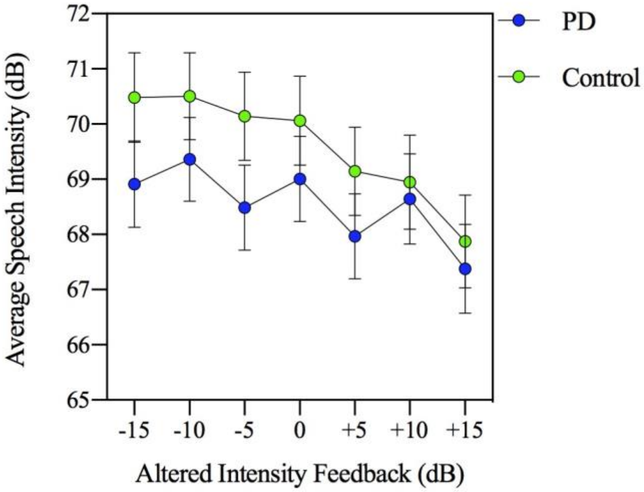


Figure 18. ***Marginal means for the PD and HC groups across 7 AIF conditions in the reading at 4x louder MP task.***

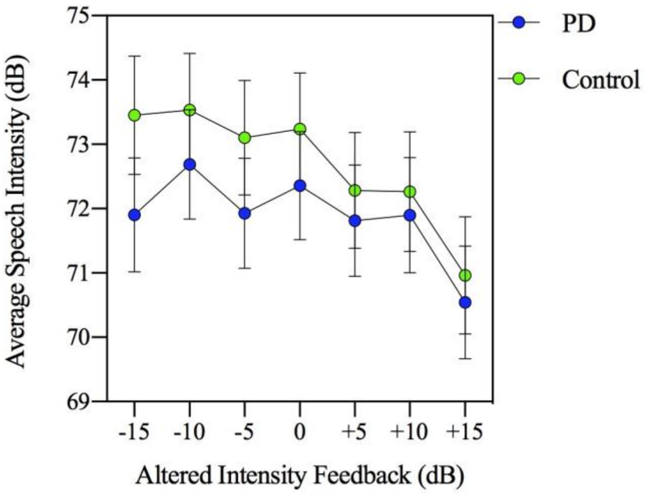
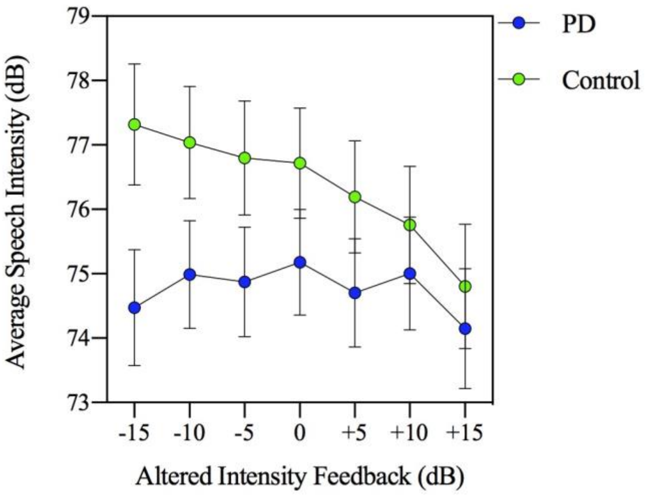


Figure 19. ***Marginal means for the PD and HC groups across 7 AIF conditions in the reading at maximum loudness MP task.***



3.4.2 The effect of background noise on the response to the MP task in PD and HC groups (Objective 5)

Objective 5 was focused on two parts. The first part was to examine the effects of background noise conditions on speech intensity in PD and HC groups. The second part was to examine the effects of the noise conditions on the response to the MP task conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the noise condition (no noise and 65dB noise) and group factors was used. In order to address part 2, a three-way ANOVA involving MP task conditions, noise conditions and group factors was used.

The descriptive statistics related to the noise conditions (no noise and 65dB background noise) for both the PD and control groups are shown in Table 16. The results of the two-way (group by background noise) ANOVA indicated that there was a significant main effect of BGN (*F* (1,48) = 28.922, *p* = 0.000). Interestingly, post hoc analysis of simple main effects revealed that the no noise condition (M = 71.67; SD = 4.50) was associated with higher speech intensity relative to the 65dB noise condition (M = 69.22; SD = 3.53) (p = .000). This result suggests that participants had more difficulty producing BGN-related increases in intensity in the context of the MP task. The group by noise condition interaction was not significant (F (1,48) = 1.149, p = .289).

Table 17. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the context of the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness).***

Background Noise	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	71.02	4.86		72.31	4.07
65 dB noise	69.06	3.86		69.38	3.13

Given this noise condition effect during the MP tasks, an important consideration was to determine if the noise conditions had a modulating effect on the MP task conditions in the PD and HC groups. In order to examine this potential modulating effect, a three-way (group by MP task conditions by noise conditions) repeated measures ANOVA for the dependent measure of speech intensity was performed. Table 17 shows the descriptive statistics for the noise conditions (no noise and 65dB background noise) during each of the four MP tasks (habitual, 2x, 4x and Max) for both the PD and control groups.

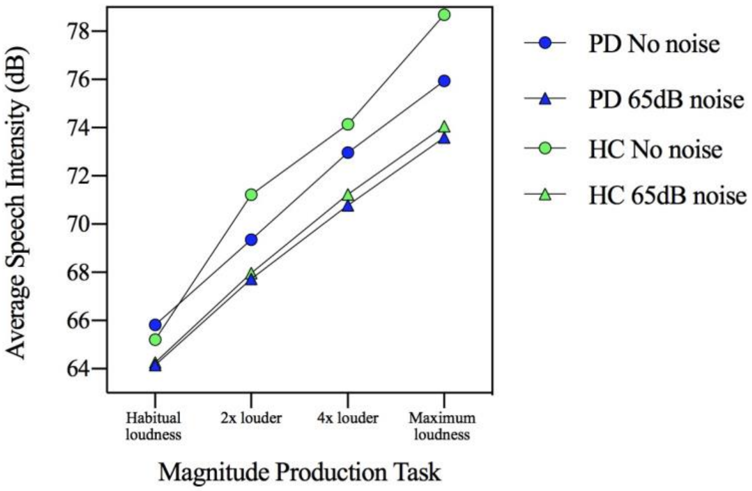
Table 18. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions and MP task obtained for the PD (n=26) and HC (n=24) groups.***

Background Noise Condition	MP Task	PD			HC	
No Noise		Mean	*SD*		Mean	*SD*
	Reading (habitual)	65.82	4.45		65.21	3.75
	2x louder	69.35	5.19		71.22	4.60
	4x louder	72.97	6.01		74.14	4.89
	Maximum loudness	75.94	5.68		78.69	4.88
65dB Noise	Reading (habitual)	64.15	4.06		64.27	2.73
	2x louder	67.72	4.27		67.97	3.26
	4x louder	70.79	4.35		71.24	3.78
	Maximum loudness	73.59	3.98		74.06	3.80

The results of the three-way ANOVA indicated that the interaction involving magnitude production task conditions, background noise conditions and groups on speech intensity was significant (*F* (3,144) = 3.715, *p* = 0.013. The three-way interaction effect is depicted in Figure 20 and illustrates how the group by noise effect modulates the group response in the MP task condition. Specifically, whereas the addition of noise caused the control group to respond differently across the MP task conditions compared to in no noise, the noise conditions do not appear to impact the pattern of speech intensity changes across the MP task conditions in the PD group.

Objective 5 focused on the effect of BGN in response to the MP task in the two participant groups, however it is noted that the 4 way interaction including the AIF levels was not significant (F (18,864) = .322, p=.997).

Figure 20. ***Marginal means for the PD and HC groups across 4 MP task conditions in no noise and 65dB background noise.***



3.5 Imitation Task (Objective 6-7)

3.5.1 Effect of imitation task on speech intensity and the response to AIF in PD and HC groups (Objective 6)

The first part of Objective 6 was to examine the effects of an imitation task on speech intensity in PD and HC groups. The second part was to examine the effects of the imitation task levels on the response to the AIF conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the imitation task levels (50dB, 60dB, 70dB, 80dB) and group factors was used.

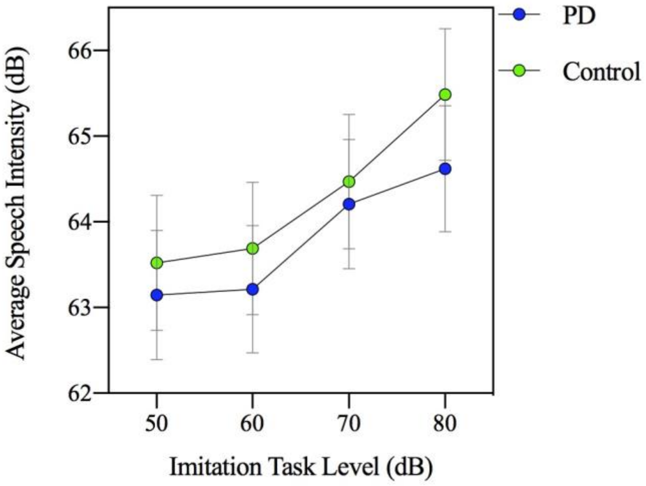
The results of the two-way (group by imitation task levels) ANOVA indicated there was a significant main effect of the imitation task (*F* (3,144) = 26.350, *p* = 0.000). The descriptive statistics related to the imitation task levels (50dB, 60dB, 70dB, 80dB) for all participants are shown in Table 18 and depicted in Figure 21. As the table and figure suggest, with the exception of the 50dB to 60dB imitation level difference, the speech intensity produced by participants increased with each successive imitation task level ($p< .05$). It is possible that either the perception of the 50dB to 60dB difference or the ability of participants to imitate sentences with high accuracy at reduced levels is more difficult. Both the main effect of group (F (1,48) = .225, p= .637) and the group by imitation task interaction (F (3,144) = .697, p = .556) were not significant.

Table 19. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 4 imitation task levels.***

Imitation Task Level			Pairwise comparisons and p values			
	Mean	*SD*	50dB	60dB	70dB	80dB
50dB	63.33	3.86				
60dB	63.45	3.79	1.000			
70dB	64.34	3.85	**.002***	**<.001***		
80dB	65.05	3.76	**<.001***	**<.001***	**.002***	

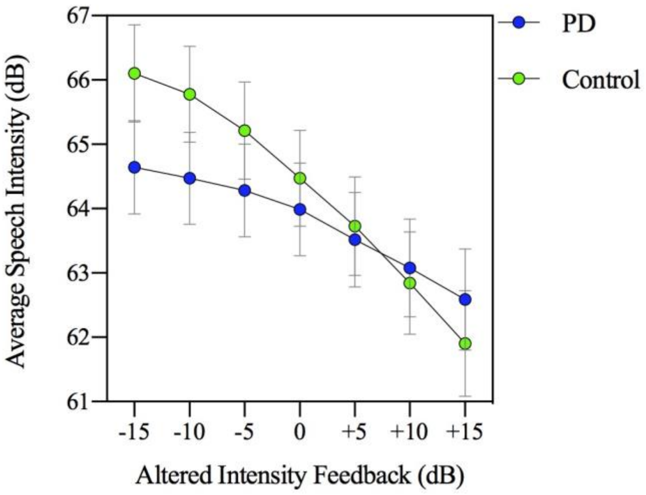
** = significant at $p < 0.05$*

Figure 21. ***Marginal means for PD and HC groups and the 4 imitation task levels.***



The second part of Objective 6 aimed to examine the potential modulating effect of the imitation task on the AIF conditions in the two groups. A three-way (group by AIF feedback condition by imitation level) repeated measures ANOVA for the dependent measure of speech intensity was performed. The three-way ANOVA results indicate the group by AIF task by imitation task interaction was not significant (F (18,864) = .919, p= .555), suggesting that the imitation levels did not have a modulating effect on the AIF conditions in the PD and HC groups. The two-way interaction involving group by AIF conditions was significant (F(6,288)= 11.317, p=.000). As depicted in Figure 22, this interaction confirms that the PD and HC groups continued to show different responses to the AIF conditions (as previously described in the results for objective 1; reduced slope by the PD group) even in the context of the 4 imitation tasks.

Figure 22. ***Marginal means for PD and HC groups and the 7 AIF conditions in the imitation task.***



3.5.2 The effect of background noise on the response to the imitation task in PD and HC groups (Objective 7)

Objective 7 was focused on the effects of different background noise conditions on speech intensity in PD and HC groups in the context of the imitation task. In order to address this 7th objective, a three-way ANOVA involving imitation task conditions, noise conditions and group factors was used.

The descriptive statistics related to the noise conditions (no noise and 65dB background noise) for both the PD and control groups are shown in Table 19. The results of the two-way (group by background noise) ANOVA revealed a significant main effect of BGN (*F* (1,48) = 16.786, *p* = 0.000). Interestingly and similar to the MP task (but dissimilar to the other speech tasks), post hoc analysis of simple main effects revealed that the no noise condition (M = 65.11; SD = 4.74) was associated with higher speech intensity relative to the noise condition (M = 62.98; SD = 3.39) (p = .000). This result suggests that participants had more difficulty producing BGN-related increases in intensity in the context of the imitation task. The group by noise condition interaction was not significant (F (1,48) = .331, p = .568).

Table 20. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the context of the imitation task (*****50dB, 60dB, 70dB, 80dB*****).***

Background Noise	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	64.71	5.26		65.50	4.11
65 dB noise	62.88	3.67		63.08	3.04

Given this noise condition effect during the imitation tasks, an important consideration was to determine if the noise conditions had a modulating effect on the imitation task conditions in the PD and HC groups. In order to examine this potential modulating effect, a three-way (group by imitation task condition by noise condition) repeated measures ANOVA for the dependent measure of speech intensity was performed. Table 20 shows the descriptive statistics for the noise conditions (no noise and 65dB background noise) during each of the four imitation tasks (50, 60, 70 and 80dB) for both the PD and control groups.

Table 21. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions and imitation task obtained for the PD (n=26) and HC (n=24) groups.***

Background Noise Condition	MP Task	PD			HC	
No Noise		Mean	*SD*		Mean	*SD*
	50dB	63.52	5.06		64.55	4.22
	60dB	64.04	5.65		64.90	4.14
	70dB	65.23	5.40		65.73	4.15

	80dB	66.05	5.52		66.83	4.49
65dB Noise	50dB	62.77	4.28		62.49	3.61
	60dB	62.39	3.79		62.49	3.18
	70dB	63.19	4.14		63.21	3.21
	80dB	63.18	3.41		64.14	2.93

The results of the three-way ANOVA indicated that the interaction involving imitation conditions, background noise and group on speech intensity was not significant (*F* (3,144) = 1.554, *p* = 0.203). These results suggest that the background noise conditions did not have a modulating effect on the speech intensity response to the 4 imitation conditions.

Objective 7 focused on the effect of the imitation task conditions in response to BGN in the two participant groups, however it is noted that the 4 way interaction including the AIF levels was not significant (F(18,864)= .820, p=.678).

3.6 Complete Masking Noise (Objective 8-10)

3.6.1 Effect of complete masking noise and speech tasks on speech intensity in PD and HC groups (Objective 8)

The aim of Objective 8 was to examine the effect of complete masking noise on speech intensity in PD and HC groups. Objective 8 was focused on two parts. The first part was to examine the effects of complete masking noise on speech intensity in PD and HC groups. The second part was to examine the effects of complete masking noise on the 4 different speech tasks in the PD and HC groups. In order to address part one, a two-way

ANOVA involving noise conditions (no noise and 100dB masking noise) and group factors was used.

The descriptive statistics related to the masking noise conditions (no noise and 100dB masking noise) for both the PD and control groups are shown in Table 21. The results of the two-way (group by masking noise condition) ANOVA indicated that there was a significant main effect of masking noise (*F* (1,47) = 320.047, *p* = 0.000). Post hoc analysis of simple main effects revealed that the 100dB masking noise condition (M = 76.40; SD = 3.11) was associated with higher speech intensity relative to the no noise condition (M = 67.59; SD = 3.37) (p = .000). The main effect of group and group by noise condition interaction were not significant (F (1,47) = .035, p = .853; F (1,47) = .061, p = .805 respectively).

Table 22. ***Descriptive statistics of marginal means and standard deviations related to the masking noise conditions obtained for the PD (n= 26) and HC (n=23) groups.***

Masking Noise Condition	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	67.57	3.56		67.60	2.48
100 dB noise	76.27	3.18		76.54	3.57

In order to examine the effect of complete masking noise on the 4 different speech tasks, a three-way ANOVA involving masking noise, speech task and group was used. The results of the three-way (group by masking noise by speech task) ANOVA indicated that there was a significant main effect of speech task (*F* (3,141) = 100.202, *p* = 0.000). The post hoc analysis of simple main effects related to the 4 speech tasks (conversation at a near distance, conversation at a far distance, vowel prolongation, and reading) are shown in Table 22. In general, the post hoc analysis of simple main effects for speech tasks revealed that speech intensity was increased in the vowel prolongation task compared to all other tasks and the sentence-reading task had lower speech intensity than all other tasks.

Table 23. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 4 speech tasks (in no noise and complete masking noise).***

Speech Task			Pairwise comparisons and p values			
	Mean	*SD*	Conversation (near)	Conversation (far)	Vowel	Reading (habitual)
Conversation (near)	71.19	3.04				
Conversation (far)	73.16	3.24	**<.001***			
Vowel	75.08	3.13	**<.001***	**<.001***		
Reading (habitual)	68.55	3.48	**<.001***	**<.001***	**<.001***	

** = significant at $p < 0.05$*

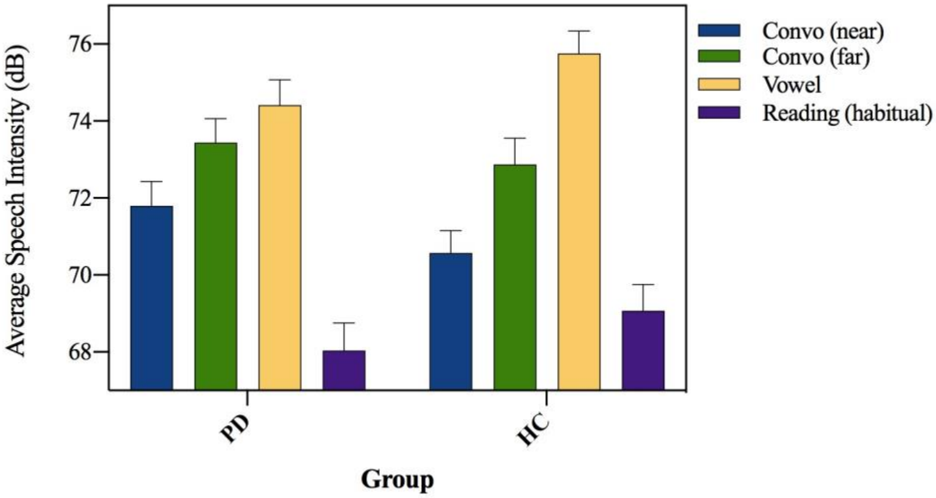
The group by speech task interaction was also significant (F (3,141) = 4.944, p = .003). The descriptive statistics related to the speech intensity obtained for the PD and HC groups during each of the speech tasks are shown in Table 23 and depicted in Figure 23.

Table 24. ***Descriptive statistics for the 4 speech tasks in the PD (n=26) and HC (n=23) groups.***

Speech Task	PD		HC	
	Mean	*SD*	Mean	*SD*
Conversation (near)	71.80	3.21	70.57	2.82
Conversation (far)	73.44	3.19	72.87	3.28

Vowel	74.41	3.37	75.75	2.82
Reading (habitual)	68.04	3.63	69.07	3.29

Figure 23. ***Marginal means for the 4 speech tasks by the PD and HC groups (no noise and 100dB masking noise).***



Post-hoc analysis involved comparisons between the PD and control groups for each of the pairwise difference scores related to the 4 speech tasks. Results of the post-hoc analysis are provided in Table 24. In general, this post-hoc analysis revealed that the group differences in speech intensity for the speech tasks was most apparent in the conversation compared to the other speech tasks (vowel and reading), and that neither the difference in conversation at near vs. far interlocutor distances nor the difference between vowel and reading speech tasks differed significantly between groups. This is consistent with the previous AIF level and slope analysis in Objective 2, confirming that the group difference is most apparent in the conversational speech tasks rather than the reading and vowel tasks and this pattern is consistent with and without complete masking noise.

Table 25. ***Interaction post-hocs involving a comparison of the two groups (PD vs. HC) for each of the 6 pairwise difference scores related to the 4 speech tasks (convN – convF), (conN – read), (convN – vowel), (convF – read), (convF – vowel), and (read – vowel) in no noise and 100dB masking noise.***

Difference Conditions	PD			HC			PD – HC difference score t-test			*p value*
	Mean	*SD*		Mean	*SD*		Mean difference	Standard error difference	t-value	
ConN - ConF	1.64	.75		2.33	1.92		-.69	.41	t(48) = -1.71	.094
ConN – Vowel	2.61	2.16		5.25	3.14		-2.64	.76	t(48) = -3.49	**.001***
ConN – Read	-3.76	2.94		-1.51	2.37		-2.25	.76	t(48) = -2.96	**.005***
ConF – Vowel	.97	2.19		2.92	3.54		-1.95	.83	t(48) = -2.36	**.022***
ConF – Read	-5.40	3.01		-3.85	2.79		-1.56	.82	t(48) = -1.89	.065
Read – Vowel	-6.37	3.58		-6.76	3.12		-.40	.95	t(48) = .41	.681

** = significant at* $p < 0.05$

The results of the three-way ANOVA also indicated that there was a significant three-way interaction involving group, masking noise condition and speech task (*F* (3,141) =9.796, *p* = 0.000). In order to interpret this three-way interaction, a separate plot of the two-way speech task by group interaction was created for each of the two masking noise conditions (no noise and 100dB masking noise). These two plots are shown in Figures 24 and 25. The descriptive statistics related to the data in these figures is presented in Table 25. Visual inspection of these two figures indicates that the group difference in the speech tasks was more pronounced during the 100dB masking noise conditions compared to in the no noise condition. In the complete masking noise condition, the control group had an increased difference in speech intensity between the conversation tasks and the vowel prolongation task and reduced difference between the conversation tasks and the reading task compared to the PD group. However, in the no noise condition, the speech intensity differences across speech tasks were similar in both groups.

Figure 24. ***Marginal means for the PD and HC groups across 4 speech tasks in the no noise condition.***

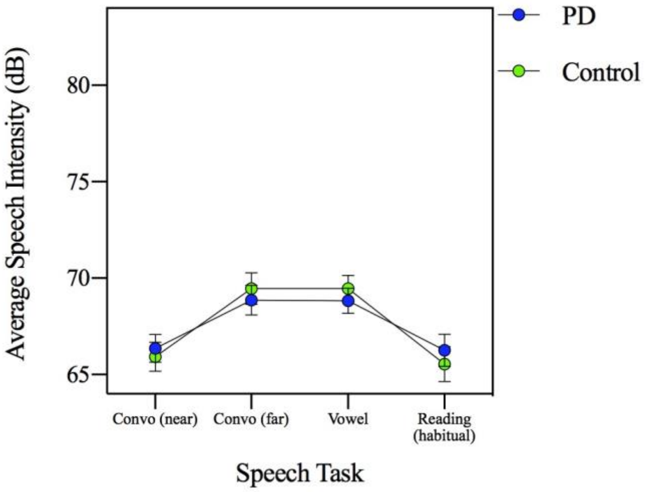


Figure 25. ***Marginal means for the PD and HC groups across 4 speech tasks in the 100dB masking noise condition.***

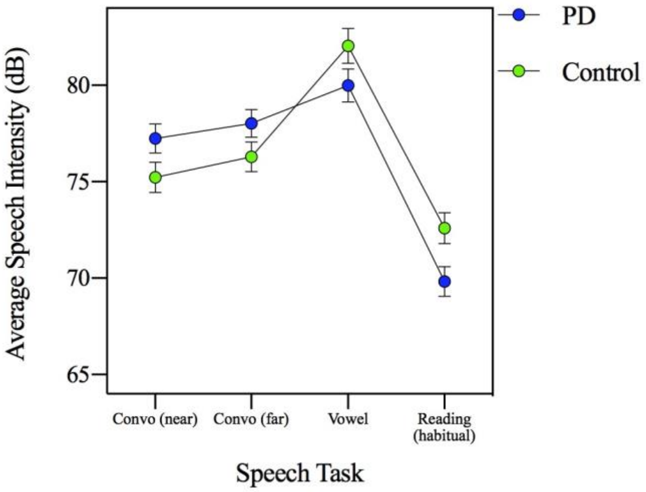


Table 26. ***Descriptive statistics for the 4 speech tasks and masking noise conditions in the PD and HC groups.***

Speech Task	Masking Noise	PD			HC	
Conversation (near distance)		Mean	*SD*		Mean	*SD*
	No noise	66.36	3.96		65.93	3.27
	100dB noise	77.24	3.76		75.22	3.82
Conversation (far distance)	No noise	68.86	4.27		69.46	3.56
	100dB noise	78.02	3.51		76.28	3.86
Vowel Prolongation	No noise	68.83	4.23		69.45	1.77
	100dB noise	79.99	3.71		82.05	4.98
Reading (habitual)	No noise	66.25	4.50		65.55	4.06
	100dB noise	69.83	3.72		72.60	4.07

3.6.2 Effect of complete masking noise and MP task conditions on speech intensity in PD and HC groups (Objective 9)

The aim of Objective 9 was to examine the effect of complete masking noise on speech intensity in PD and HC groups. Objective 9 was focused on two parts. The first part was to examine the effects of complete masking noise on speech intensity in PD and HC groups in the context of the MP task. The second part was to examine the effects of complete masking noise on the 4 different MP task conditions in the PD and HC groups. In order to address part one, a two-way ANOVA involving noise conditions (no noise and 100dB masking noise) and group factors was used.

The descriptive statistics related to the masking noise conditions (no noise and 100dB masking noise) for both the PD and control groups are shown in Table 26. The results of the two-way (group by masking noise condition) ANOVA indicated that there was a significant main effect of masking noise (*F* (1,48) = 21.208, *p* = 0.000). Post hoc analysis of simple main effects revealed that the 100dB masking noise condition (M = 74.54; SD = 3.27) was associated with higher speech intensity relative to the no noise condition (M = 72.11; SD = 4.41) (p = .000). The main effect of group (F (1,48) = 2.071, p = .157) and group by noise condition interaction were not significant (F (1,48) = .155, p = .695 respectively).

Table 27. ***Descriptive statistics of marginal means and standard deviations related to the masking noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the MP task.***

Masking Noise Condition	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	71.52	4.76		72.70	3.97
100 dB noise	73.75	2.74		75.34	3.76

In order to examine the effect of complete masking noise on the 4 MP task conditions, a three-way ANOVA involving masking noise, MP task and group was used. The results of the three-way (group by masking noise by MP task) ANOVA indicated that there was a significant main effect of MP task (*F* (3,144) = 260.754, *p* = 0.000). The post hoc analysis of simple main effects related to the 4 MP tasks conditions (habitual loudness, 2x louder, 4x louder, maximum loudness) are shown in Table 27. In general, the post hoc analysis of simple main effects for the MP task revealed that speech intensity increased with each successive MP loudness task. The group by MP task interaction was not significant (F (3,144) = 1.148, p = .332).

Table 28. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 4 MP task conditions (in no noise and complete masking noise).***

Magnitude Production Task			Pairwise comparisons and p values			
	Mean	*SD*	Habitual loudness	2x louder	4x louder	Maximum loudness
Habitual loudness	68.48	3.48				
2x louder	72.47	3.83	**<.001***			
4x louder	74.80	3.80	**<.001***	**<.001***		
Maximum loudness	77.56	3.68	**<.001***	**<.001***	**<.001***	

** = significant at* $p < 0.05$

The results of the three-way ANOVA also indicated that there was a significant three-way interaction, involving group, masking noise condition and MP task (F (3,144) =6.617, p = 0.000). In order to interpret this three-way interaction, a separate plot of the two-way MP task by group interaction was created for each of the two masking noise conditions (no noise and 100dB masking noise). These two plots are shown in Figures 26 and 26. The descriptive statistics related to this data is presented in Table 28.

Visual inspection of these two figures indicates that while in the no noise condition the group difference is most apparent in the higher MP task conditions (4x louder, maximum loudness), in the complete masking noise, the group difference is most apparent in the lower MP task conditions (habitual loudness, 2x louder).

Figure 26. ***Marginal means for the PD and HC groups across 4 MP task conditions in the no noise condition.***

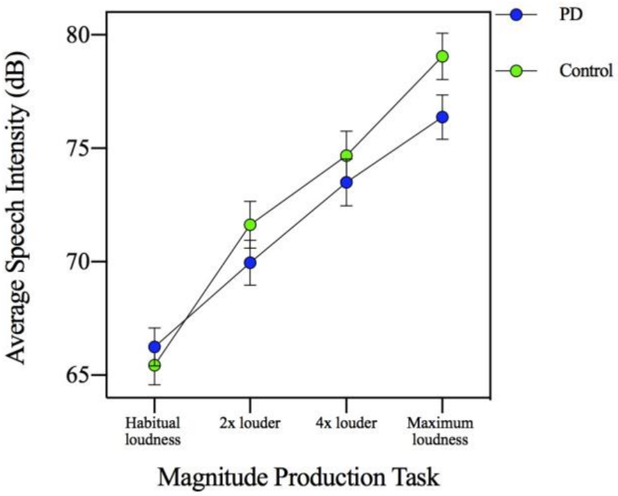


Figure 27. ***Marginal means for the PD and HC groups across 4 MP task conditions in the 100dB masking noise condition.***

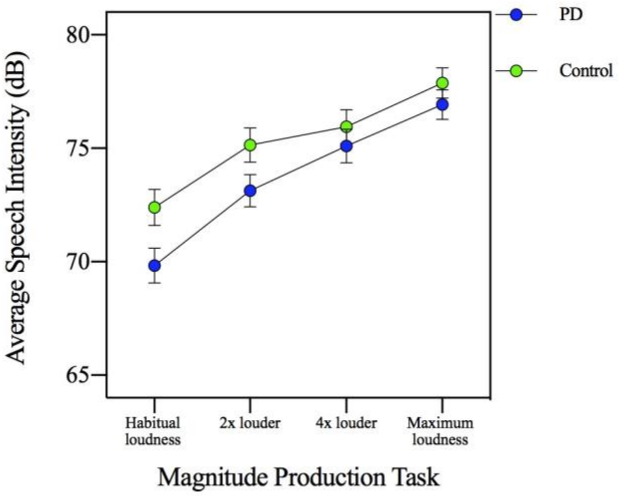


Table 29. ***Descriptive statistics for the 4 MP task conditions and masking noise conditions in the PD (n=26) and HC (n=24) groups.***

Magnitude Production Task	Masking Noise	PD			HC	
Habitual Loudness		Mean	*SD*		Mean	*SD*
	No noise	66.25	4.50		65.44	4.01
	100dB noise	69.83	3.72		72.40	4.10

2x Louder	No noise	69.96	5.42		71.63	4.64
	100dB noise	73.13	3.22		75.14	4.08
4x Louder	No noise	73.50	5.64		74.67	4.90
	100dB noise	75.10	2.81		75.94	4.52
Maximum Loudness	No noise	76.37	5.45		79.05	4.50
	100dB noise	76.93	2.46		77.87	4.01

3.6.3 Effect of complete masking noise on speech intensity and performance on the intensity imitation task in PD and HC groups (Objective 10)

The aim of Objective 10 was to examine the effect of complete masking noise on speech intensity and the performance on the intensity imitation tasks in PD and HC groups. The first part of Objective 10 was to examine the effects of complete masking noise on speech intensity in PD and HC groups. The second part was to examine the effects of complete masking noise on the 4 different intensity imitation task conditions in the PD and HC groups. In order to address part one, a two-way ANOVA involving noise conditions (no noise and 100dB masking noise) and group factors was used.

The descriptive statistics related to the masking noise conditions (no noise and 100dB masking noise) for both the PD and control groups are shown in Table 29. The results of the two-way (group by masking noise condition) ANOVA indicated there was a significant main effect of masking noise (*F* (1,48) = 76.474, *p* = 0.000). Post hoc analysis of simple main effects revealed that the 100dB masking noise condition (M = 70.55; SD = 4.66) was associated with higher speech intensity relative to the no noise condition (M = 65.26; SD = 3.87) (p = .000). The main effect of group (F (1,48) = 2.736, p = .105) and group by noise condition interaction were not significant (F (1,48) = 2.417, p = .127 respectively).

Table 30. ***Descriptive statistics of marginal means and standard deviations related to the masking noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the imitation task.***

Masking Noise Condition	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	64.86	5.17		65.66	4.03
100 dB noise	69.21	3.63		71.89	4.11

In order to examine the effect of complete masking noise on the 4 imitation task conditions, a three-way ANOVA involving masking noise, imitation task and group was used. The results of the three-way (group by masking noise by imitation task) ANOVA indicated that there was a significant main effect of imitation task (*F* (3,144) = 34.375, *p* = 0.000). The post hoc analysis of simple main effects related to the 4 imitation tasks conditions (50dB, 60dB, 70dB, 80dB) are shown in Table 30. In general, the post hoc analysis of simple main effects for the imitation task revealed that speech intensity increased with each successive imitation loudness condition. The group by imitation task interaction was not significant (F (3,144) = .324, p = .808).

Table 31. ***Post-hoc results related to pairwise comparisons involving the marginal means for the 4 imitation task conditions (in no noise and complete masking noise).***

Imitation Task			Pairwise comparisons and p values			
	Mean	*SD*	50dB	60dB	70dB	80dB
50dB	66.80	3.73				
60dB	67.51	3.93	**.023***			

70dB	68.21	3.97	**<.001***	**<.001***		
80dB	69.10	3.76	**<.001***	**<.001***	**<.001***	

* = *significant at* $p < 0.05$

The results of the three-way (group, masking noise condition and imitation task) ANOVA were not significant (F (3,144) = 0.568, p = .637).

3.7 Instruction to Ignore Auditory Feedback (Objective 11-12)

3.7.1 Effect of instructions to ignore auditory feedback on speech intensity and the response to AIF in PD and HC groups (Objective 11)

Objective 11 was focused on two parts. The first part was to examine the effects of the instruction to ignore the auditory feedback on speech intensity in PD and HC groups. The second part was to examine the effects of the instruction to ignore feedback task on the response to the AIF conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the instruction to ignore conditions (reading at habitual loudness with no instructions, reading with instruction to ignore the auditory feedback and maintain a constant speech intensity) and group factors was used.

The results of the two-way (group by instruction conditions) ANOVA indicated that there was a significant main effect of the instruction condition (F (1,48) = 57.927, p = 0.000), and indicated that speech intensity produced by participants when asked to ignore the auditory feedback (M=61.82; SD= 3.39) was significantly reduced compared to when asked to read at a habitual loudness with no instructions regarding the auditory feedback (M= 64.86; SD= 3.38, p= .000). Both the main effect of group (F(1,48)= .157, p= .694), and the group by instruction condition interaction (F (1,48) = .056, p = .814) were not significant.

Given there was an effect of the instruction condition on speech intensity, the second part of Objective 11 was to examine the potential modulating effect of this instruction condition on the AIF conditions in the two groups. A three-way (group by AIF feedback condition by instruction condition) repeated measures ANOVA for the dependent measure of speech intensity was performed. The three-way ANOVA results indicate the group by AIF task by instruction condition interaction was not significant (F (6,288)= .382, p= .890). This is depicted in Figures 28 and 29 such that the slope of the PD and control group are similar across the two instruction conditions however the overall intensity is reduced in the instruction to ignore auditory feedback condition (Figure 29). The AIF by group interaction was significant (F(6, 288)= 5.315, p=.000), consistent with previous findings (reduced slope of the PD group). This is depicted in Figure 30.

Figure 28. ***Marginal means for PD and HC groups and the 7 AIF conditions in the reading with no instruction task.***

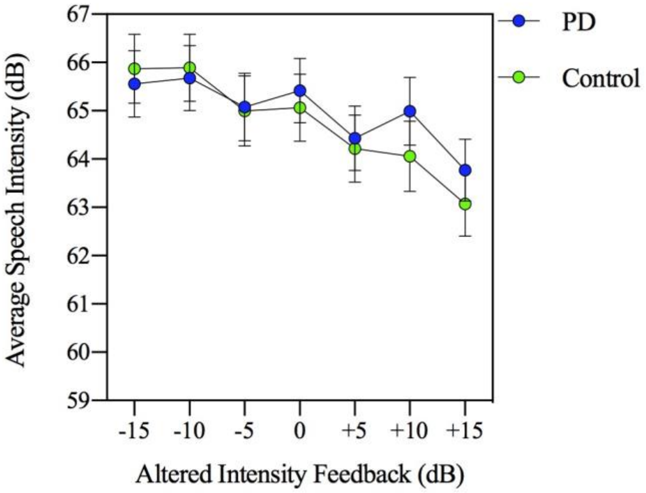


Figure 29. ***Marginal means for PD and HC groups and the 7 AIF conditions in the reading with an instruction to ignore auditory feedback task.***

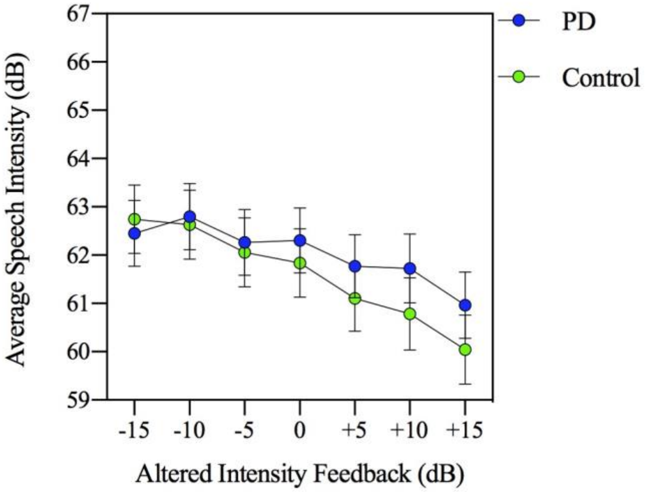
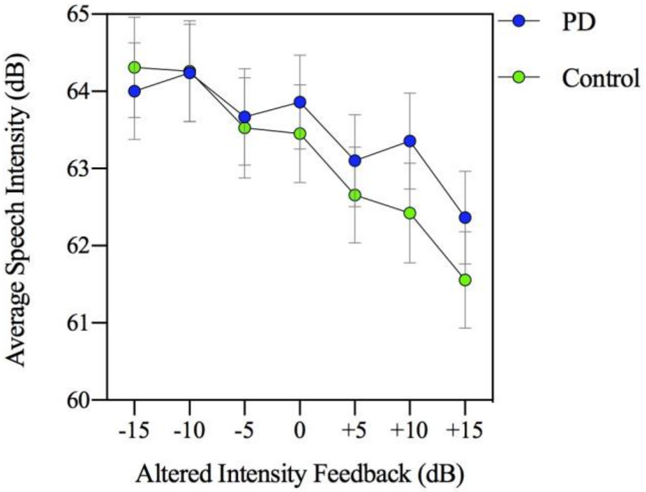


Figure 30. ***Marginal means for PD and HC groups and the 7 AIF conditions in the instruction to ignore auditory feedback conditions (combined with and with no instruction).***



3.7.2 The effect of the instruction conditions on the response to background noise conditions in PD and HC groups (Objective 12)

Objective 12 was focused on the effect of different background noise conditions on speech intensity in PD and HC groups in the context of the instruction conditions. In order to address this objective, a three-way ANOVA involving instruction conditions, noise conditions and group factors was used. The results of the three-way ANOVA indicated that there was no significant main effect of the noise conditions (*F* (1,48) = .950, *p* = 0.334), and no significant three-way interaction (F (1,48) = .439, p = .511). These results suggest that in the context of the instruction conditions, the noise condition did not impact the speech intensity produced by the PD or control group. Descriptive statistics related to this result are presented in Table 31.

Table 32. ***Descriptive statistics of marginal means and standard deviations related to the background noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the instruction to ignore auditory feedback conditions (with and without instruction).***

Background Noise Condition	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	63.45	3.73		62.92	2.93
65 dB noise	63.58	3.38		63.41	2.92

3.8 Self-Loudness Perception (Objective 13-17)

3.8.1 Self-loudness perception ratings of speech intensity in the context of the MP Task and the response to AIF in PD and HC groups (Objective 13)

Loudness perception ratings were obtained during 3 of the 7 AIF levels (-10dB, 0dB, +10dB) during the MP task. Participants were asked to indicate how loud they perceive their own speech (self-loudness rating) by placing a dash along a visual analogue scale line (endpoints labeled low loudness and high loudness). Measurement of these ratings was collected in millimetres (mm). In order to examine these ratings across the two groups, objective 13 was focused on two parts. The first part was to examine the loudness perception ratings during the Magnitude Production (MP) tasks in PD and HC groups. The second part was focused on the examination of self-loudness perception ratings during the MP task while also experiencing the AIF conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the MP levels (habitual loudness, 2 times louder, 4 times louder, maximum loudness) and group factors was used.

The results of the two-way (group by MP task levels) ANOVA indicated that there was a significant main effect of the MP task (F (3,144) = 48.002, p = 0.000). The descriptive statistics related to the loudness perception ratings in the MP task for all participants are shown in Table 32 and depicted in Figure 31. As the table and figure suggest, the loudness perception ratings by participants increased with each successive magnitude production level (p<.001). The main effect of group was found to be significant (F(1,48)= 4.665, p= .036). Interestingly, the PD group was observed to have higher self-loudness ratings (M=61.09; SD= 16.93) compared to the control group (M=53.62; SD= 17.62). This higher self-loudness value is contrary to the lower speech intensity values that were found in the MP task (and all other speech tasks). To emphasize this potentially important difference between the PD participants' perceived self-loudness and their actual speech intensity, Figure 32 is a re-presentation of Figure 14 from Objective 4 to allow for a visual comparison alongside the self-loudness figure (Figure 31) of the self-loudness ratings and speech intensity values for the MP task.

Table 33. ***Post-hoc results related to pairwise comparisons of loudness perception ratings (mm on a 100mm visual analogue rating scale) involving the marginal means for the 4 MP levels.***

Magnitude Production Level		Pairwise comparisons and p values

	Mean	*SD*	Habitual	2x louder	4x louder	Maximum
Habitual	47.32	10.51				
2 x louder	56.30	12.64	**<.001***			
4 x louder	60.86	14.53	**<.001***	**.001***		
Maximum	64.94	17.15	**<.001***	**<.001***	**.001***	

** = significant at $p < 0.05$*

Figure 31. ***Loudness perception marginal means for PD and HC groups and the 4 MP task levels.***

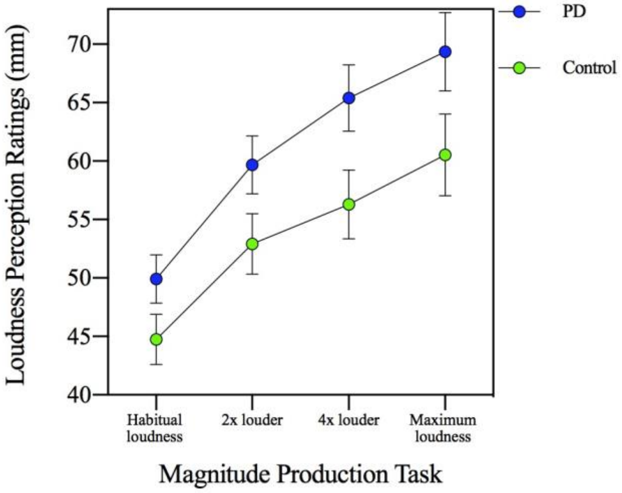
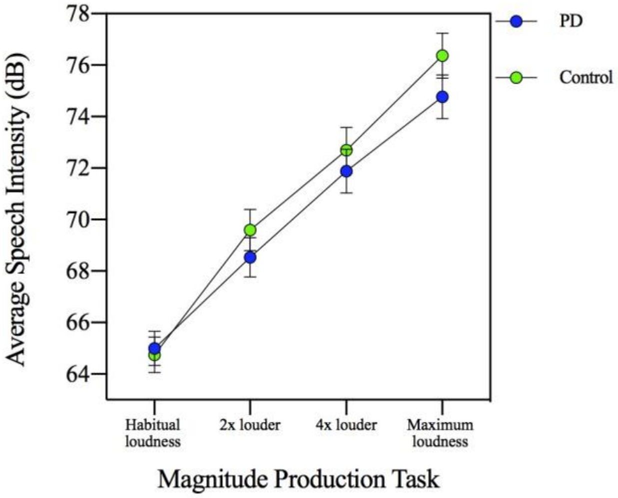


Figure 32 (14). ***Marginal means for PD and HC groups and the 4 MP task levels.***



The group by MP task interaction was not significant (F (3,144) = .717, p = .543). Another interesting finding was that the two-way interaction involving group by AIF conditions was not significant (F (2,96)=2.039, p=.136). Therefore, the PD and control groups had similar loudness perception ratings across the different AIF conditions, despite consistently showing significantly different speech intensity responses in all other objectives. Figure 33 (loudness perception ratings) and Figure 34 (modified Figure 15 from Objective 4), highlight the distinction between the speech intensity responses and the loudness perception ratings to AIF in the two groups.

Figure 33. ***Mean loudness perception ratings for PD and HC groups and the 3 AIF levels in the MP task.***

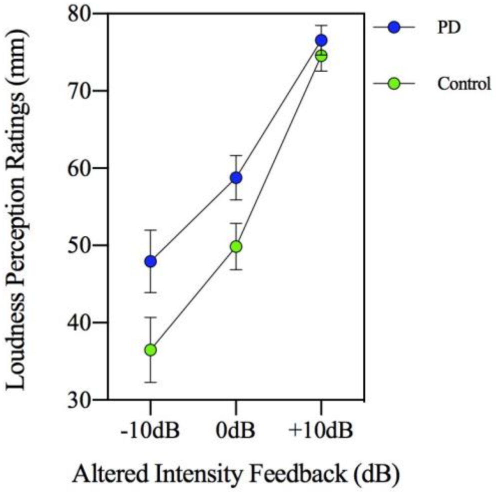
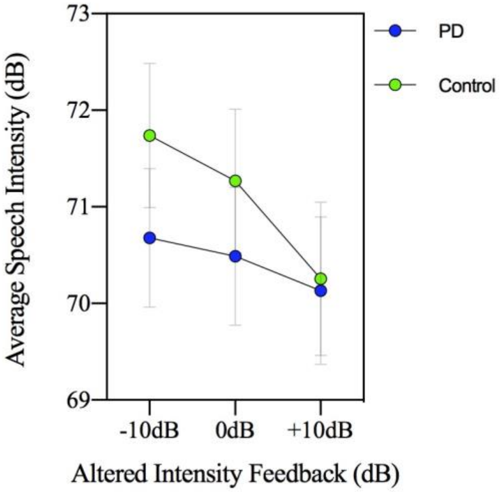


Figure 34 ***(Modified Figure 15). Mean speech intensity for PD and HC groups and the 3 AIF levels in the MP task.***



Given there was an effect of the MP task on loudness perception ratings, the second part of Objective 13 aimed to examine the potential modulating effect of this MP task on the AIF conditions in the two groups. A three-way (group by AIF feedback condition by MP level) repeated measures ANOVA for the dependent measure of loudness perception rating was performed. The three-way ANOVA results indicate the group by AIF task by MP task interaction was significant (F (6,288)= 2.288, p= .036), suggesting that the MP levels had a modulating effect on the AIF conditions in the PD and HC groups. This three-way interaction is depicted in Figures 35, 36, 37, and 38 and descriptive statistics are provided in Table 33. It appears this significant interaction is a result of the loudness perception in the 4x loudness and maximum loudness MP conditions. As depicted in Figure 37, 38 and Table 33, the control group produced a steeper slope across the AIF levels compared to the PD group in these conditions. This steeper slope of loudness ratings across AIF levels by the control group is in contrast to the relatively consistent flatter slope of loudness ratings by the PD group.

Figure 35. ***Loudness perception marginal means for the PD and HC groups across 3 AIF conditions in the reading at habitual loudness MP task.***

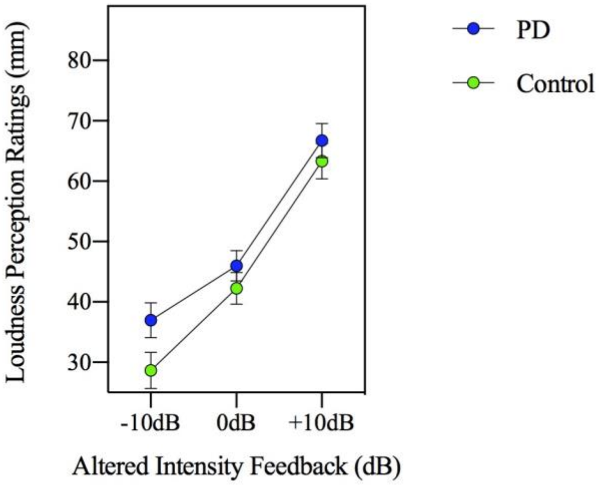


Figure 36. ***Loudness perception marginal means for the PD and HC groups across 3 AIF conditions in the reading at 2x louder MP task.***

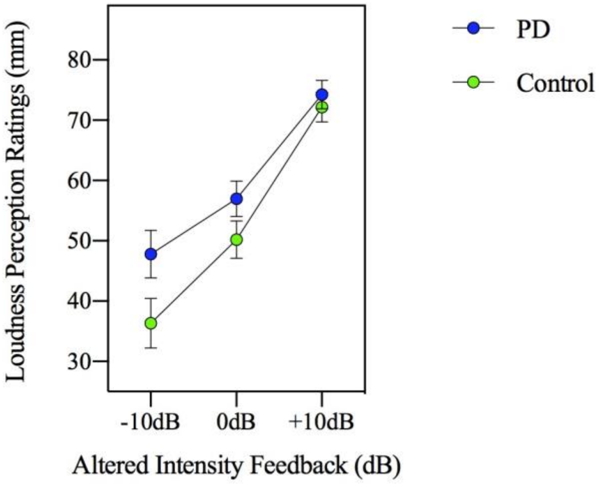


Figure 37. ***Loudness perception marginal means for the PD and HC groups across 3 AIF conditions in the reading at 4x louder MP task.***

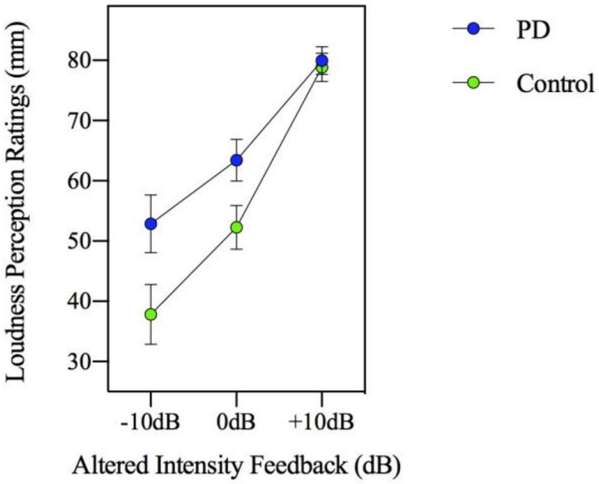


Figure 38. ***Loudness perception marginal means for the PD and HC groups across 3 AIF conditions in the reading at maximum loudness MP task.***

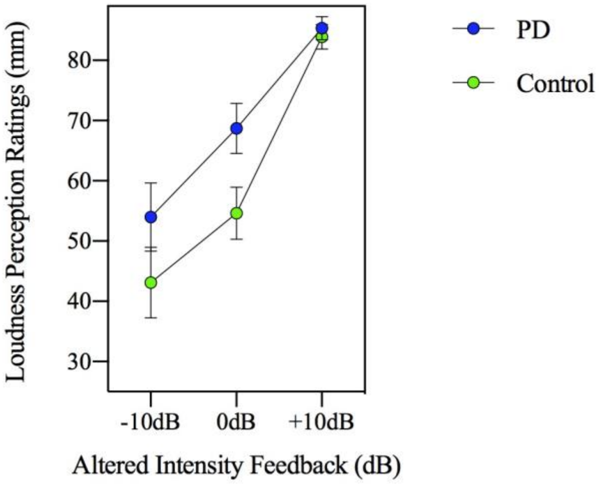


Table 34. ***Descriptive statistics for loudness perception ratings in the 4 MP task levels and AIF levels in the PD and HC groups.***

Magnitude Production Task	AIF Level	PD			HC	
Habitual		Mean	*SD*		Mean	*SD*

Loudness	-10dB	36.99	15.28		28.64	14.15
	0dB	46.01	14.59		42.27	10.96
	+10dB	66.73	13.68		63.30	15.03
2x Louder	-10dB	47.82	22.48		36.35	17.49
	0dB	56.95	13.75		50.22	16.64
	+10dB	74.27	10.96		72.18	13.34
4x Louder	-10dB	52.87	25.28		37.81	23.30
	0dB	63.40	18.04		52.28	17.74
	+10dB	79.95	10.18		78.82	13.00
Maximum Loudness	-10dB	54.01	31.06		43.09	26.19
	0dB	68.68	20.69		54.60	21.83
	+10dB	85.36	8.20		83.90	11.38

3.8.2 The effect of background noise on self-loudness perception ratings in the MP task by PD and HC groups (Objective 14)

Objective 14 was focused on two parts. The first part was to examine the effects of a background noise conditions on self-loudness perception ratings in PD and HC groups. The second part was to examine the effects of the noise conditions on the self-loudness ratings during the MP task conditions in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the noise condition (no noise and 65dB noise) and group factors was used. In order to address part 2, a three-way ANOVA involving MP task conditions, noise conditions and group factors was used.

The descriptive statistics related to the loudness perception ratings in the noise conditions (no noise and 65dB background noise) for both the PD and control groups are shown in Table 34. The results of the two-way (group by background noise) ANOVA indicated that there was a significant main effect of noise (*F* (1,48) = 4.583, *p* = 0.037). Post hoc analysis of simple main effects revealed that the no noise condition (M = 58.56; SD = 12.77) was associated with higher ratings of perceived loudness relative to the 65dB noise condition (M = 56.15; SD = 12.94) (p = .037). These perceptual rating results are consistent with the measures of speech intensity produced in noise conditions from Objective 5. The group by noise condition interaction was not significant (F (1,48) = 1.129, p = .293).

Table 35. ***Descriptive statistics of loudness perception rating marginal means and standard deviations related to the background noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the context of the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness).***

Background Noise	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	62.89	12.30		54.23	13.24
65 dB noise	59.28	13.80		53.01	11.90

Given this noise condition effect during the MP tasks, an important consideration was to determine if the noise conditions had a modulating effect on the loudness ratings in the MP task conditions in the PD and HC groups. In order to examine this potential modulating effect, a three-way (group by MP task conditions by noise conditions) repeated measures ANOVA for the dependent measure of loudness perception ratings was performed. This three-way ANOVA was not significant (*F* (3,144) = 1.198, *p* = 0.313.

Objective 14 focused on the effect of noise conditions in response to the MP task on loudness ratings in the two participant groups, however it is noted that the 4 way interaction including the AIF levels was not significant (F(6,288)= .434, p=.856).

3.8.3 Self-loudness perception ratings of speech intensity in the context of the instructions to ignore auditory feedback and the response to AIF in PD and HC groups (Objective 15)

Loudness perception ratings were also obtained during 3 of the 7 AIF levels (-10dB, 0dB, +10dB) in the context of the instructions to ignore auditory feedback. In order to examine these ratings across the two groups, Objective 15 was focused on two parts. The first part was to examine the loudness perception ratings of the instructions to ignore conditions (with and without instructions) in PD and HC groups. The second part was to examine the loudness perception ratings of speech intensity in the instructions conditions in the context of the AIF levels in the PD and HC groups. In order to address part 1, a two-way ANOVA involving the instructions conditions (no instruction, with instruction) and group factors was used.

The results of the two-way (group by instruction conditions) ANOVA indicated that the main effect of instruction condition (*F* (1,48) = 2.110, *p* = 0.153) and the group by instruction condition interaction (*F* (1,48) = .043, *p* = 0.836) were not significant.

The two-way interaction involving group by AIF conditions was not significant (F (2,96)= .162, p=.850), Thus, (similar to in the MP task), the PD and control groups had similar loudness perception ratings across the different AIF conditions, despite showing significantly different speech intensity responses in the different instruction conditions.

3.8.4 The effect of background noise on self-loudness perception ratings in the instruction to ignore auditory feedback conditions by PD and HC groups (Objective 16)

Objective 16 was focused on the effect of different background noise conditions on loudness ratings in PD and HC groups in the context of the instruction conditions. In order to address this objective, a three-way ANOVA involving instruction conditions, noise conditions and group factors was used.

The main effect of noise conditions (F(1,48)=.058, p=.811), noise by group interaction (F(1,48)=.329, p=.569), and three-way (instruction by noise by group) interaction (F(1,48)= .002, p=.966) were not significant.

3.8.5 Self-loudness perception ratings of speech intensity in the context of complete masking noise in the MP task (Objective 17)

Self-loudness perception ratings were also obtained during the MP task in the context of complete masking noise (100dB background noise). Objective 17 was focused on two parts. The first part was to examine the effects of complete masking noise on loudness perception ratings in PD and HC groups. The second part was to examine the effects of complete masking noise on the self-loudness ratings obtained during the MP task conditions in the PD and HC groups. In order to address part one, a two-way ANOVA involving noise conditions (no noise and 100dB masking noise) and group factors was used. In order to address part 2, a three-way ANOVA involving MP task conditions, noise conditions and group factors was used.

The results of the two-way (group by noise conditions) repeated measures ANOVA indicated the main effect of noise conditions was not significant (F(1,48)=2.618, p=.112). Descriptive statistics are provided in Table 35. This result suggests that the participants rated their speech loudness as similar whether in no noise or in complete masking noise despite producing a significantly increased speech intensity in the complete masking noise condition compared to the no noise condition (objective 8). The main effect of group (*F* (1,48) = 2.089, *p* = 0.155) and the group by noise condition interaction (*F* (1,48) = 3.298, *p* = 0.076) were not significant. It should be noted that although this interaction was not significant, the control group rated their speech as louder in the complete masking noise whereas the PD group did not.

A three-way ANOVA (MP task by noise conditions by group) was used to examine the loudness perception ratings during the MP tasks when combined with the masking noise conditions. The main effect of MP task was significant (*F* (3,144) = 92.760, *p* = 0.000). The descriptive statistics related to the loudness perception ratings in the MP task for all participants in the two noise conditions are shown in Table 36 and depicted in Figure 39. As the table and figure suggest, the loudness perception ratings by participants increased with each successive magnitude production level (p<.000). This is consistent with the speech intensity levels from Objective 9. The three-way ANOVA results indicate the group by noise conditions by MP task interaction was not significant (F (3,144)= 2.364, p= .074).

Table 36. ***Descriptive statistics of loudness perception rating marginal means and standard deviations related to the complete masking noise conditions obtained for the PD (n= 26) and HC (n=24) groups in the context of the MP task (habitual loudness, 2x louder, 4x louder, maximum loudness).***

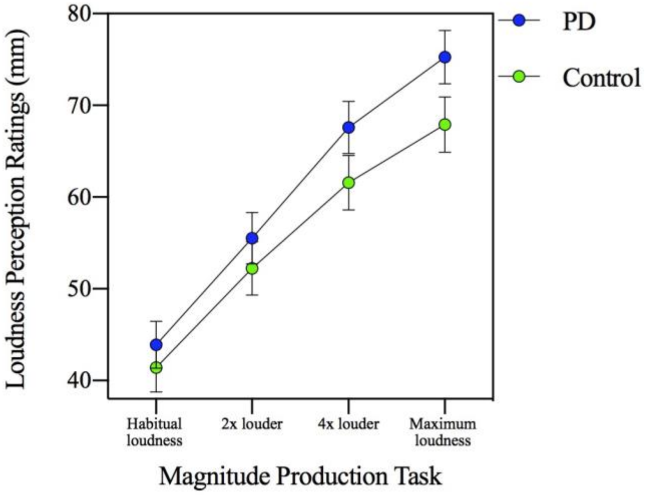
Complete Masking Noise	PD			HC	
	Mean	*SD*		Mean	*SD*
No noise	60.89	14.36		50.15	16.42
100 dB noise	60.24	17.14		61.41	17.82

Table 37. ***Post-hoc results related to pairwise comparisons of loudness perception ratings involving the marginal means for the 4 MP levels in the context of the noise conditions (no noise and 100dB noise).***

Magnitude Production Level			Pairwise comparisons and p values			
	Mean	*SD*	Habitual	2x louder	4x louder	Maximum
Habitual	42.66	13.03				
2 x louder	53.87	14.34	**<.001***			
4 x louder	64.58	14.56	**<.001***	**.001***		
Maximum	71.58	14.82	**<.001***	**<.001***	**.001***	

** = significant at $p < 0.05$*

Figure 39. ***Loudness perception marginal means for PD and HC groups and the 4 MP task levels in the context of the noise conditions (no noise and 100dB masking noise).***



Chapter 4: Discussion

Sensorimotor integration deficits have been hypothesized as an explanation for several of the clinical symptoms associated with PD including hypokinesia and bradykinesia (Bronstein et al., 1990; Klockgether & Dichgans, 1994; Rinalduzzi et al., 2015)). Previous research suggests that the speech problems in PD may be related to abnormal auditory perception or auditory-motor integration processes. Despite the work conducted on speech intensity perception and production, there remains a paucity of literature addressing this issue in the context of a range of communicative situations and speech tasks. Error correction tasks enable the study of this potentially dysfunctional system in PD. Thus, the current study examined the impact of altered intensity feedback on speech intensity regulation in PD. The aim of this study was to provide descriptions of the response to AIF in the context of a range of communicative tasks and conditions as the regulation of speech intensity may vary depending on the communication environment and communicative goals. Speech tasks and varying degrees of communicative goals and the effects they may have on speech intensity are not always predictable but are identified as having potential effects on intensity in PD. So, the current study sought to examine the effects of different speech tasks on intensity and the response to altered auditory feedback during different speech tasks.

In addition, two different communicative environments were included because of their well-known effects on intensity level. These conditions included interlocuter distance and background noise. Increases in interlocuter distance and the level of background noise are consistently associated with increases in speech intensity. These conditions were also examined during altered intensity feedback conditions in order to better understand the role of auditory feedback during these intensity-modulating conditions.

Two different intensity production tasks were included to examine the voluntary production of self-estimated intensity levels and the reproduction of external intensity levels. The self-estimated intensity production task was a magnitude production task (i.e. 2x louder, 4x louder, maximum loudness) and the reproduction of external intensity levels task was an imitation task (i.e. target sentences at 50dB, 60dB, 70dB, and 80dB). The effect of these intensity production tasks on speech intensity were examined in isolation and when combined with altered intensity feedback.

A self-loudness perceptual rating procedure was included in the study in order to examine the participants' self-perception of loudness during the condition and task related changes in speech intensity. An important aspect of this part of the study is the comparison of self-loudness perception and actual speech intensity production.

The use of altered auditory feedback for speech intensity may involve voluntary or involuntary control. An instruction to ignore the altered auditory feedback condition was included to examine the participants' ability to deliberately ignore the altered auditory feedback and maintain a constant loudness of their voice. This task was completed with and without background noise.

Another way to examine the extent to which speech intensity is regulated by auditory feedback is by measuring speech production in complete masking noise. This condition involved presenting 100dB of background noise to the speaker and analyzing their speech intensity in the various communication tasks.

Through this wide range of speech tasks and speaking conditions, a group of participants with PD was examined to further elucidate the possible abnormal sensorimotor integration deficit related to speech production.

The next part of the discussion will be organized around each of the study objectives. These objectives will be discussed in detail so as to provide the primary findings as well as evidence-based explanations for each specific objective. This will be followed by a summative discussion of the findings from this study with interpretations related to our understanding of the role of auditory feedback for speech intensity control in PD, limitations of the current study, as well as the broader clinical implications.

4.1 Altered Intensity Feedback (AIF)

4.1.1 Effect of AIF on Speech Intensity (Objective 1)

Objective 1 aimed to examine how individuals with PD use auditory feedback for speech intensity regulation. In particular, this objective examined how PD participants respond to AIF and how these responses impact speech intensity regulation. This objective was achieved by analyzing the response to the 7 AIF levels in the context of 4 different speech tasks (conversation near and far, vowel prolongation, reading sentences).

In the current study, all participants (PD and control) displayed a presumed compensatory response to the AIF levels such that as AIF levels increased, the speech intensity of participants decreased and vice versa. However, the response to AIF was different between the two groups. Specifically, the slope of the AIF function was significantly reduced in the PD group. In addition, the magnitude of the response to AIF was significantly reduced in the PD group compared to the control group. This reduced magnitude of the response to AIF in the individuals with PD was observed in both the positive and negative directions of AIF. This is in contrast to previous studies that used an auditory perturbation paradigm. In perturbation studies, responses to very brief (~200ms) random shifts in auditory feedback are examined typically in the context of prolonged vowels. These studies found larger magnitudes of compensation produced by PD groups (Chen et al., 2013; Huang et al., 2016; Mollaei et al., 2013, Mollaei et al., 2016). However, these studies perturbed vocal pitch and formant frequencies and it is possible that speech intensity regulation involves different sensorimotor processes than pitch and formant frequency regulation. Liu and colleagues (2012) however, found larger response magnitudes in their PD participants with intensity perturbations. It is also possible that the perturbation paradigm involves different feedback control mechanisms compared to AIF as the former involves very brief alterations. Some researchers suggest the compensations observed in perturbation paradigms are involuntary or reflexive in nature, because these speakers are unable to suppress the response and they occur without the speakers’ intent (Abur et al., 2018; Zarate & Zatorre, 2008). However, other studies discuss the possibility of a complex response to auditory perturbations such that depending on the latency of the response, it may be either involuntary (100-150ms) or voluntary (250-600ms) (Patel et al., 2014). Future PD studies are recommended that examine the same speech tasks and conditions to compare the perturbation and AIF paradigms. In addition, examination of response latency in the AIF paradigm is recommended for future studies to further examine the possibility of voluntary and involuntary responses.

Another interesting finding from this objective is that when the negative (-15dB, -10dB, -5dB) and positive (+15dB, +10dB, +5dB) directions were compared, the magnitude of the compensation response was significantly less in the negative direction (-15dB in both groups). This suggests either a possible reduced sensitivity to decreased loudness (resulting in a smaller compensation response in this direction) or a greater sensitivity to increases in loudness (resulting in a larger compensation response in this direction). It is also possible that this is reflective of a reduced relative importance of decreased loudness of speech to the system such that mechanisms to control for increased loudness are more “primed” for regulation as only louder speech has the potential to be damaging and uncomfortable to the speaker.

Overall, the results from Objective 1 provide insight into the use of auditory feedback for speech intensity regulation in the PD group. Although all participants produced speech intensity that opposed the direction of the AIF, the PD group’s reduced response is indicative of abnormal integration of auditory feedback for speech intensity production. Based on these findings, it is suggested that PD speakers either have abnormal perception of the intensity of their speech or they are unable to appropriately integrate the auditory information of their speech for motor execution.

4.2 Speech Tasks

4.2.1 Effect of Different Speech Tasks on Speech Intensity and the Response to Altered Intensity Feedback (Objective 2)

The aim of Objective 2 was to examine whether different types of speech tasks would impact the speech intensity of the participants and also the response to AIF of the participants. Previous research suggests that individuals with PD produce increased speech intensity during speech tasks that do not have clear communicative goals, such as vowel phonation, syllable repetition, and sentence reading (quasi-speech tasks) compared to monologue tasks (Ramig, Countryman, O'Brien, Hoehn, & Thompson, 1996; Ramig & Dromey, 1996; Fox & Ramig, 1997). In addition, unlike control participants who show an automatic adjustment of their speech in conversational samples by increasing their intensity, PDs display a greater reduction in speech intensity during conversational tasks (Ho et al., 1999; Winkworth & Davis, 1994). However, in the current study, both participant groups produced higher speech intensity and a steeper slope of the AIF function in the conversational speech tasks relative to the sentence-reading task. The nature of the conversation task may be contributing to the unique findings in the current study. Specifically, the conversation task in the current study involved a dialogue between the participant and the experimenter, which may be a different experience compared to the 20-30 second monologue task used in previous studies (Hansen & Boril, 2018). The higher conversational speech intensity and steeper AIF slope in the current study may be related to the increased communicative demand of conversing with another person compared to reading a sentence, such that the speech motor planning system places a greater priority on intelligible speech while conversing. Further, the conversation task, in the context of increased interlocutor distance, led to increased speech intensity in both groups. This finding is consistent with previous interlocutor distance studies in non-neurologically impaired participants (Cheyne et al., 2009; Traunmüller & Eriksson, 2000) and in PD participants (Adams et al., 2010; Ho et al., 1999; McCaig et al., 2015).

In the context of the AIF paradigm, group differences related to the different speech tasks emerged. Particularly, the group difference in the AIF slope function was most pronounced in the conversation task relative to the vowel prolongation and reading tasks. In other words, the PD group produced significantly reduced compensations to the altered feedback specifically in the context of having a conversation. This was in comparison to the PD's similar responses compared to the control group in both the vowel and reading tasks. Adams and Dykstra (2009) hypothesized that the compounded attentional demands associated with a conversation task may have an impact on speech intensity regulation. This may provide an explanation as to why these speech task differences were observed. Based on this hypothesis, a difference in the PD response to the reading task compared to the conversation task is expected, because a reading task is presumably less demanding of attentional resources. Specifically, if increased attentional demands are forcing the PD group to produce reduced compensations in the conversation task, then increased responses to the AIF (in comparison to the conversation task) are expected in the reading task. It is important to note, however, that responses to the AIF by the PD group in conversation (far distance) and the reading task were similar in the current study (Figure 9). Thus, alternative explanations for the more apparent reduced response by the PD group in the conversation tasks are warranted.

A communicative-goal hypothesis is suggested. The increased communicative goals or demands associated with the conversation task provide a possible explanation. Perhaps we engage in different feedback processes or place increased priority on auditory feedback of our own voice when engaged in speech tasks requiring clear communicative goals and greater communicative demands. It is possible that in PD, either this increased priority is not engaged for cognitive reasons (e.g. Theory of Mind), or subcortical mechanisms and subcortical/cortical pathways are disrupted such that this feedback monitoring-motor execution process is not appropriately initiated or is excessively inhibited. Future studies are recommended that systematically manipulate attentional demands (e.g. cognitively demanding dual tasks) and speech tasks with varying communicative intent to further elucidate the current findings and explanations.

The current study expands on previous work by Ho and colleagues (1999) who found that individuals with PD failed to adjust their volume (positive direction AIF level testing only) in a conversation task, and results from studies of altered feedback on quasi-speech tasks (syllable, reading, and counting tasks), which found that PD participants respond similarly to controls (Brajot et al., 2016; Coutinho et al., 2009; Ho et al., 1999). Interestingly, although the current study found reduced compensations by the PD group in the conversation tasks, the difference between responses to AIF in the different interlocutor conditions by the PD group was similar to the control group. Thus, the PD group does not display an overt deficit in distance judgment as it pertains to conversing with a listener, which may be suspected if a further reduction was observed in the far interlocutor distance condition. Rather, the PD group displayed an overall disruption in the regulation of speech intensity and abnormal use of altered auditory feedback in all conversation tasks. The current study suggests that individuals with PD have abnormal processing of auditory information for speech intensity regulation, and this disruption particularly impacts their ability to regulate speech intensity in the context of speech tasks with clear communicative goals (i.e. conversational speech).

4.3 Background Noise

4.3.1 Effect of Background Noise on Speech Intensity and the Response to Altered Intensity Feedback (Objective 3)

Objective 3 aimed to examine the effect of different background noise conditions on speech intensity and whether the noise conditions would affect the AIF response. Consistent with previous studies (Adams et al., 2006; Garnier et al., 2010; Ho et al., 1999; Lane & Tranel, 1971; Pick et al., 1989) the presentation of background noise was found to elicit an increase in speech intensity (i.e. Lombard response) in both groups (PD and controls). Individuals with PD-related hypophonia have been shown in previous studies to display an “overall gain reduction” for speech intensity and a gradually decreasing signal-to-noise ratio with increasing background noise (Adams et al., 2006; Ho et al., 1999; Iulianella et al., 2008). PD participants in the current study were not observed to produce speech intensity at a reduced level compared to the controls in the background noise condition. It is possible that a reduced intensity was not observed in the current study PD group due to the variance and/or hypophonia severity levels of the participants.

The abnormal response to AIF in the PD group observed in objective 1 appeared to be differently affected by the background noise. Specifically, although the PD group produced a flatter slope in the AIF response than the controls in no noise, in the context of 65dB background noise, the group difference was emphasized (PD group was observed to produce a much flatter slope of the AIF function compared to the control group). It appears that when individuals with PD are speaking in a noisy environment, abnormal sensorimotor integration for speech intensity regulation is more pronounced. It is possible this is a result of a reduced range of speech intensity production capacity (Clark et al., 2014; De Keyser et al., 2016). However evidence from the MP task in Objective 5 indicates that the PD group in the current study had a larger speech intensity range capacity (64.15dB-75.94dB; 11.79dB range) compared to the range that was utilized in these speech tasks with background noise (67.58dB-69.55dB; 1.97dB range). Therefore, when the environmental condition requires a change in speech intensity, the range of available speech intensity or the intensity capacity is not being appropriately engaged.

The Lombard effect has been shown in a wide range of non-human animals and evidence suggests that the primary neural mechanisms for this response are subcortical (for a review see Luo, Hage, and Moss, 2018). However other studies have demonstrated that humans have a certain degree of control over the response and therefore a volitional neural network is also proposed (Luo et al., 2018; Patel & Schell, 2008). Similar to the speech task effect observed in Objective 2, the group differences in this Objective may be related to the reduced ability of the PD group to appropriately engage mechanisms in tasks with clear communicative goals. In the control group, the background noise may be eliciting a feedback monitoring process that is distinct from that used in the no noise condition due to the fact that speech intelligibility is at risk of being compromised in noise; a communicative-goal hypothesis as it relates to the Lombard response. In fact, previous studies have considered this as a possible explanation for the Lombard effect, such that this reflex is engaged so as to mediate reduced speech intelligibility and maintain clarity of speech when communicating (Patel & Schell, 2008).

Overall, the current study suggests that the abnormal processing of auditory information for speech intensity regulation observed in PD may be particularly pronounced when speaking in noisy environments.

4.4 Magnitude Production (MP) Task (Objective 4-5)

4.4.1 Effect of MP Tasks on Speech Intensity and the Response to Altered Intensity Feedback (Objective 4)

The aim of Objective 4 was to examine the effects of a Magnitude Production (MP) task on speech intensity and to determine if the MP task would modulate the response to AIF.

MP tasks require a scaling of speech intensity across productions. This task is inherently complex, as it requires the speaker to perceive the loudness of their voice, estimate a comparatively higher level of self-loudness, and accurately perform the motor output to achieve the intended loudness. This task therefore involves deliberate self-estimation and self-monitoring of speech production with a greater degree of focus on internal targets relative to other speech tasks (i.e. conversation, imitation tasks) and the MP task may require less external guidance or focus than other speech tasks such as imitation. Overall, participants in the current study were observed to successfully complete the task and successively increase the intensity of their speech across MP task conditions. The current study is consistent with work by Dromey and Adams (2000), and did not find a significant difference between PD and control participants. In contrast, a previous study by Clark and colleagues (2014) found a flatter slope of the loudness function in their PD participants. However Clark and colleagues (2014) examined a wider range of MP task conditions (i.e. 2 additional soft conditions; 2x and 4x softer), and the flatter slope found in their study may be attributed to these additional conditions. It is worth noting, however, that although the difference between groups did not reach significance in the present study, the PD participants were observed to produce a slightly flatter slope of the MP response than the controls.

Interestingly, the previously observed flatter PD slope of the AIF function obtained for the Objective 1 speech tasks (conversation, vowel prolongation, reading at habitual loudness) was found to remain flatter in the PD group during the MP conditions as well. Thus, despite the MP task involving deliberate self-monitoring of speech intensity, the PD group continued to show an abnormality in their use of auditory feedback to regulate their speech intensity. This is important because both groups displayed successive increases in speech intensity across the MP task levels. Therefore, it may be suggested that the PD group is using an alternate method in order to monitor and make these successive increases in speech intensity. This also suggests that a scaling ability is present in the PD group. As previously discussed, the MP task is a more internally focused speech task and external feedback may not play a large role. If PD speakers have a particular deficit in the processing of external feedback for motor control (i.e. excessive inhibition), perhaps the highly internal focus of the MP task is why they are generally more successful in achieving a similar MP function to controls. In contrast, the overall gain setting was abnormal in the PD group (overall reduced loudness compared to controls), but this initial gain setting may be less reliant on internal targets and rather an external focus is required. It is possible that the PD group is unable to use external information for appropriate gain setting.

The near significant three-way interaction involving group, MP task, and AIF levels provided insight into the use of auditory feedback for speech regulation in the MP task. Results suggest that control participants made presumed compensations to the auditory feedback changes in an overall consistent manner across the different MP task conditions. However, the slope of the AIF function produced by the PD participants became increasingly flatter as the MP task loudness requirements increased. In other words, for the PD group, the task requirements of producing louder speech resulted in a more pronounced reduction in the use of auditory feedback for speech intensity regulation. This does not appear to be the result of a ceiling effect in speech intensity capacity in the PD group, as higher intensity responses were observed in the complete masking noise condition (M= 76.93dB; SD=2.46). However, the maximum loudness condition of the MP task in the context of AIF is an entirely different experience for the speaker compared to in the context of masking noise. For example, with positive AIF levels (+5dB, +10dB, +15dB) the speaker is increasing their loudness while simultaneously receiving auditory feedback of speech that is even louder, compared to in complete masking and an absence of auditory feedback altogether. Perhaps the combination of effects in the maximum loudness condition and AIF, made it even more difficult for the PD group to use the external feedback appropriately.

4.4.2 Effect of Background Noise on the Response to the MP Task (Objective 5)

Objective 5 was aimed at examining the effect of different background noise conditions on speech intensity in the MP task and whether the noise conditions would affect the AIF responses in this task. Although the Lombard response is typically elicited in background noise, and this was observed in the context of the other speck tasks (conversation, vowel prolongation, reading), this response was not elicited in the context of the MP task. The current study results indicate reduced speech intensity in background noise and this is inconsistent with observations of the Lombard response in previous studies (Adams et al., 2006; Garnier et al., 2010; Ho et al., 1999; Lane & Tranel, 1971; Pick et al., 1989). It is possible that the AIF levels contributed to this unexpected result, however analysis of the 0dB AIF level yielded a similar effect of reduced speech intensity in background noise. To our knowledge, only one previous study has looked at the effect of background noise in the context of a MP task. Clark (2012) did not directly compare the noise conditions, however their data are suggestive of an increase in speech intensity in the control group from no noise (62.50-76.70dB MP task range) to the 65dB background noise condition (68.45-79.92 MP task range). The noise condition difference in the PD group was 60.89-71.10dB MP task range in no noise to 67.99-75.45dB MP task range in 65dB background noise. Thus it appears that both the PD and control group showed a minimum 3dB increase in speech intensity in the 65dB noise condition during the MP task. Further studies are required to investigate whether a Lombard response is anticipated across MP task conditions in the context of noise, and possible reasons that this response was not triggered in the current study.

A previous study examined the ability to suppress the Lombard response, and suggested that tasks in which increased attention towards speech intensity are possible, enable a suppression of the Lombard response (Pick et al., 1989). It is possible that since the MP task requires specific directed attention towards speech intensity, that Lombard suppression occurs. In contrast, it is also possible that the additional cognitive/attentional demands of the MP task produce an “overloaded” system and the appropriate speech intensity modulations are not engaged. Alternatively, the communicative-goal hypothesis as it relates to the Lombard response may apply here as well. The Lombard effect is susceptible to cortical control and increased communicative goals result in increased Lombard responses (Garnier et al., 2010; Patel & Schell, 2008). Perhaps the MP task involves reduced communicative intent and so the Lombard response is not elicited. It is also possible that the internal focus of the MP task produced a reduced Lombard response similar to what was observed in the PD group in response to the AIF during this task.

The significant three-way interaction (group by noise condition by MP task) indicates that the pattern of speech intensity changes across the MP task conditions were less impacted in the PD group. Specifically, the control participants produced a steeper slope of the loudness function (rate of speech intensity increase across successive increases in MP task condition levels) in no noise, while the background noise condition resulted in a flatter slope of the function, however, the PD group was observed to produce speech similarly in both noise and no noise. Thus, a similar postulation is provided such that the MP task may be causing the control group to suppress the Lombard response. In contrast, the MP task does not appear to impact the Lombard response in the PD group to the same degree.

4.5 Imitation Task (Objective 6-7)

4.5.1 Effect of Imitation Task on Speech Intensity and the Response to Altered Intensity Feedback (Objective 6)

Imitation tasks provide a unique opportunity to examine sensorimotor integration of speech intensity because in order to accomplish this task the individual must process the auditory information, plan, and execute a corresponding speech intensity level. All participants produced increases in speech intensity with increasing target intensity levels. The PD group produced imitations that were reduced in comparison to those of the control group, however this difference was not statistically significant. These findings are consistent with a study by Adams and colleagues (2006). Other studies of imitation tasks by PD participants have shown reduced target imitation levels compared to controls (Clark et al., 2014; De Keyser et al., 2016), however De Keyser and colleagues found the group differences in the 80dB imitation level only. Thus, further research is recommended to determine the degree to which and the conditions under which performance deficits in imitation tasks may be observed in the PD population. It is possible that the AIF conditions had an influence on the results in the current imitation task.

Interestingly, the reduced (flatter) response to the AIF levels in the PD group was also present in the context of the imitation task. The imitation task itself involves complex processing across multiple speech-related systems. The participant is first required to attend to externally generated stimuli (4 different intensity levels), plan a comparable internally generated intensity, produce the planned imitation with accuracy, and monitor the intensity of their speech throughout the production. Therefore, this task requires attentional/planning/cognitive demands, external perceptual demands, internal intensity demands, and self-monitoring processes. This complex sequence of demands coupled with the unique demands of the AIF paradigm, may have caused the PD group to respond differently to the AIF compared to the control group. It is possible the control group is better able to manage the complexities associated with combining the imitation task with the AIF manipulations.

Overall, the reduced response to AIF in this task indicates that the group difference in the AIF effect is consistent across several tasks and conditions and therefore seems to be quite robust.

4.5.2 Effect of Background Noise on the Response to the Imitation Tasks (Objective 7)

The aim of Objective 7 was to examine the effect of background noise on the performance of the imitation task. Similar to the results from the MP task, the addition of 65dB of noise in the imitation task resulted in reduced speech intensity by both groups. The absent Lombard effect may again be explained by a possible communication goal hypothesis. It is possible that the Lombard was suppressed due to the lack of clear communication goals in the imitation task. It is also possible that the task of modulating speech intensity in the context of noise, forces the speaker to suppress the Lombard reflex in order to accomplish the target intensity. These hypotheses are possible, since previous studies have indicated the relative ease of voluntarily suppressing the Lombard response in the context of a reading task (less demanding task) compared to in conversation/monologue (Vinney, van Mersbergen, Connor, & Turkstra, 2016; Garnier et al., 2010). Future studies are suggested to examine perceptual ratings of speaking effort in the context of AIF and background noise in a range of tasks in order to examine this in more detail.

4.6 Complete Masking Noise (Objective 8-10)

4.6.1 Effect of Complete Masking Noise on Speech Intensity during Different Speech Tasks (Objective 8)

The role of auditory feedback for speech intensity regulation is unclear and the AIF paradigm used in the current study is one way of examining this in more detail. Another way of examining the degree to which speakers rely on auditory perception for speech production is by blocking the auditory feedback entirely. Objective 8 focused on determining the effect of speech intensity in a range of speaking tasks (conversation, vowel prolongation and reading sentences) when a speaker is exposed to complete masking of their auditory feedback. To our knowledge, this is the first study to examine the effect of complete masking noise on speech intensity in a range of speech tasks. Analysis of speech intensity when speaking without the ability to monitor auditory feedback may provide information about the importance of auditory feedback.

The current study found that both individuals with PD and control participants increased the intensity of their speech while speaking in masking noise. It is possible that the Lombard effect contributed to this increase in intensity. Future studies could examine responses to increasing levels of background noise to determine the level at which a change in speech production occurs and differentiates the Lombard response from the response to complete masking. In addition, future studies could examine alternate ways to examine the role of masked auditory feedback in speech intensity regulation such as deaf speakers with and without cochlear implants.

Consistent with findings from Objective 2, the control group was observed to produce speech intensity that was very different across the different speech tasks such that conversation tasks were found to be distinct from the reading and vowel tasks. When the difference in each speech task was examined in complete masking noise condition, the speech task effect became even more pronounced. This suggests that task-related differences in the communicative goal became more apparent when auditory feedback was removed in the control group. In other words, it is suggested that the control group was able to produce speech intensity that was reflective of differences in communicative goals even when auditory feedback was completely absent. In contrast, the PD group produced speech intensity that did not reflect this type of communicative intent distinction and the complete masking noise had a relatively small impact on the differences across speech tasks. Therefore, it is suggested that in the absence of auditory feedback, the control group was able to emphasize and prioritize communicative goal distinctions, whereas the PD group did not produce speech intensity that was reflective of these same distinctions.

4.6.2 Effect of Complete Masking Noise on Speech Intensity during the Performance of the MP Task (Objective 9)

Objective 9 focused on determining the effect of complete masking noise in the context of the MP task. As previously discussed, the MP task involves creating an internal representation and scaling the production of speech intensity across different loudness levels. The ability to do so, in the absence of auditory feedback, is important to understand and may provide insight into the relative importance of external auditory feedback and the degree of internal focus during this task. Although both groups were able to complete the MP task appropriately and increase their loudness with each successively louder MP condition (whether in no noise or in complete masking), an interesting finding emerged when the complete masking noise condition was examined. It was observed that the control group produced a steeper slope across the MP conditions in the no noise condition and a flatter slope in the masking noise condition. This suggests that the absence of auditory feedback in the MP task disrupted the control group's ability to scale the loudness of their speech.

This is in comparison to the PD group, to whom the masking noise had little impact on their performance in the MP task. Therefore, in the context of an MP task produced in a no noise condition, control speakers may have a primarily internal focus, however there is a degree of feedback monitoring that occurs and is required in order to scale their loudness and without this feedback (i.e. complete masking), the appropriate scaling of loudness across the MP conditions is disrupted. In comparison, the current results suggest that the PD group do not utilize auditory feedback when completing a MP task and therefore the complete masking of auditory feedback had no effect on their performance of the MP task.

4.6.3 Effect of Complete Masking Noise on Speech Intensity during the Performance of the Imitation Task (Objective 10)

The aim of this Objective was to examine the impact of complete masking noise on performance in the imitation task. As previously discussed, the imitation task involves complex processes including attentional factors, planning, and integrating both internal and external information in order to attempt to imitate the 4 different target intensity levels.

Participants in both groups were found to increase their speech intensity in the complete masking noise condition. Unlike the flatter slope observed in complete masking during the MP task by the control group, the complete masking during the imitation task did not impact the slope of the imitation function in either participant group. This suggests that although there is some disruption in the ability to imitate target intensities, the unique processes involved in maintaining the relative differences across increasing levels of speech intensity stimuli are less dependent on auditory feedback. It is possible that because the imitation task involves a combination of external focus (externally generated stimuli) as well as internal predictions, the participants are better able to re-produce a heard intensity even when auditory feedback of their own voice is blocked. In other words, it is possible that speakers are better able to scale the loudness of their speech when an externally generated model is provided (as opposed to an internally generated model, as in the MP task).

4.7 Instruction to Ignore Auditory Feedback (Objective 11-12)

4.7.1 Effect of Instruction to Ignore Auditory Feedback on Speech Intensity (Objective 11)

The degree to which a speaker has control over the use of auditory feedback for speech intensity regulation is unknown. Objective 11 aimed to examine the effect of an instruction to ignore the auditory feedback on speech intensity. Results suggest that the response to AIF levels was not impacted by the instruction to ignore auditory feedback. The participants in the current study were unable to voluntarily regulate the intensity of their speech in the context of AIF and maintain a constant loudness. Instead, they responded in a similar way across the AIF conditions as they did without any explicit instructions. The only difference was that the participants in both groups reduced the overall intensity of their speech in the instruction to ignore feedback condition. It is presumed that this observed reduction was caused as the participants attempted to regulate intensity. Perhaps the added attentional/cognitive demands of this task are causing a reduced intensity in a similar manner as observed in the effect of background noise on the MP task and imitation task (Objectives 5 and 7). It is possible that the instruction to ignore task forces participants to have an internal focus on the loudness of their speech and the complexity of this task impacts the overall ability to maintain a typical loudness of speech. Still, the impact of the AIF levels was difficult to ignore for these participants and similar slopes of the AIF function were observed with and without instruction to ignore. Thus, the altered intensity feedback effect was robust with the control group showing a steeper slope while the PD group consistently showing a reduced slope of the AIF function across most tasks and conditions.

It appears that when auditory feedback is available, the speaker is unable to voluntarily ignore this and focus on maintaining a constant loudness based on other speech mechanisms. This difficulty could be due to the saliency of auditory feedback, the overreliance on this type of feedback, or the under reliance on other types of mechanisms for monitoring speech intensity. Future studies should consider methods to distinguish between these possible causes. For example, a potential study could examine the possibility that alternate mechanisms for monitoring speech intensity are not being automatically engaged, but if externally cued, these mechanisms could be used to ignore auditory feedback with greater success. Alternate cues could be provided to participants to assist with maintaining their loudness such as a visual cue using a sound level meter.

4.7.2 Effect of Background Noise on the Response to the Instruction to Ignore Auditory Feedback (Objective 12)

This Objective aimed to examine the effect of background noise on the ability to ignore auditory feedback. Results suggest that speech intensity was not impacted by the addition of background noise and the results by both groups were similar. This suggests that, similar to previous noise-related discussions in earlier sections, it appears that the attentional demands and/or communicative demands of the task may be working to suppress the Lombard effect in these participants. The additional attentional demands required for focus on internal targets and/or the reduced communicative demands of this reading task are potentially reducing the Lombard effect.

4.8 Self-loudness Perception (Objective 13-17)

4.8.1 Self-Loudness Perception Ratings of Speech Intensity in the Context of the MP Task and the Response to AIF (Objective 13)

This Objective aimed to examine the self-loudness perception ratings of all participants in the MP task. All participants rated their speech as successively louder with each successive MP condition. Of interest, the PD group was observed to rate the loudness of their speech as being louder compared to the control group despite the PD group producing reduced speech intensity. Consistent with previous studies of loudness perception in PD, the current study found that individuals with PD have an inaccurate perception of their self-generated speech loudness (Clark et al., 2014; Ho et al., 1999; Ho et al., 2000; De Keyser et al., 2016; Kwan & Whitehill, 2011) and overestimate their loudness.

Further, with regard to the AIF levels, although the PD group produced a flatter slope of the function in the MP task, they nevertheless rated the loudness of the speech similarly to how control participants rated their loudness. Thus, these results suggest that the PD group also have an inaccurate perception of the scaling of their loudness. In other words, as the AIF levels increased and the actual speech intensity of the PD group decreased very minimally (flat slope of the AIF function), they nevertheless perceived their speech to be louder and continued to overestimate their loudness.

Although the following did not reach significance (approached), the PD group was observed to produce a flatter speech intensity slope of the AIF function specifically in the maximum loudness MP condition. The PD loudness ratings did not accurately match this speech intensity finding. In fact, the PD loudness ratings remained fairly consistent (flatter slope) across all MP conditions. The previously discussed primarily internal focus of the MP task may help to explain these findings. If the PD group is over-reliant on internal targets in this task (possibly related to deficits in their external feedback system), then these internal targets are the basis of their estimations and inaccurate overestimations of loudness may be expected. In other words, the PD group may be relying on their internal targets for their loudness ratings and consequently their ratings are based on their expectations of produced loudness rather than on the external auditory information of their actual productions.

4.8.2 Effect of Background Noise on Self-Loudness Perception Ratings of Speech Intensity in the Context of the MP Task (Objective 14)

This Objective aimed to examine the effect of background noise on loudness perception ratings in the MP task. The loudness ratings in background noise were consistent with the intensity produced; all participants rated their speech as louder in the context of no noise and quieter in noise. This finding suggests that the internal focus of the MP task may be guiding these judgments. The participants may have based their loudness ratings on the anticipated target of loudness they were aiming to achieve in each condition.

The PD group displayed a reduced slope across increasing MP task loudness conditions, however their ratings remained similar to the control group. The internal focus of the MP task may also explain why the differences in speech intensity were observed between groups and across MP conditions however the ratings between groups remained similar. These findings support the previous literature suggesting that individuals with PD have an inaccurate perception of the loudness of their own voice (Clark et al., 2014; Ho et al., 1999; Ho et al., 2000; De Keyser et al., 2016; Kwan & Whitehill, 2011). The current study expands on previous findings and proposes that PD participants overestimate the loudness of their speech, and these inaccuracies are present in the context of altered auditory feedback.

4.8.3 Self-Loudness Perception Ratings of Speech Intensity in the Context of the Instruction to Ignore Auditory Feedback and the Response to AIF (Objective 15)

This Objective aimed to examine the loudness perception ratings while participants were asked to ignore the auditory feedback of their speech and maintain a constant loudness. Since participants were asked to rate their speech loudness while being instructed to maintain a constant loudness level, is important to note that in the context of this task, participants were essentially completing an accuracy rating of their ability to maintain their loudness.

The loudness ratings in this task were similar across groups and similar whether in the no instruction condition or the instruction condition. This is consistent with the findings of similar speech intensity produced in these two conditions. This is also consistent with anecdotal evidence during data collection. While completing the instruction to ignore auditory feedback task, participants in both groups noted that they believed they were keeping their loudness constant through the study (including when no instructions to ignore feedback were provided). Therefore, the similarity in loudness perception ratings were to be expected. This also means that participants perceived their efforts to ignore auditory feedback were accurate.

With regard to the AIF levels, although the PD group produced a flatter slope of the AIF intensity function, they nevertheless rated the loudness of their speech similarly to control ratings. These results are consistent with findings from other Objectives in the current study that suggest the PD group had an inaccurate perception of their speech loudness/intensity. In other words, as the AIF levels increased and the actual speech intensity of the PD group decreased very minimally (flat slope of the AIF function), they nevertheless overestimated the loudness of their speech.

4.8.4 Effect of Background Noise on Self-Loudness Perception Ratings of Speech Intensity in the Context of the Instruction to Ignore Auditory Feedback (Objective 16)

It was important to determine if the addition of background noise would impact the loudness perception ratings during the instruction to ignore auditory feedback condition. Consistent with the actual speech intensity produced in noise during this task, the loudness perception ratings were not different than the perception ratings in no noise. These findings suggest that the instruction to ignore auditory feedback task was difficult for the participants to accomplish with accuracy however they believed they were able to complete the task appropriately. The participants may have been using internal targets (and ignoring external auditory feedback) to perform the reading task. It is suggested that participants in this study may have the false impression that their internal targets are reliable sources of information. These results also suggest that whether loudness ratings are made in noise or no noise, the perception of loudness is unchanged.

4.8.5 Effect of Complete Masking Noise on Self-Loudness Perception Ratings of Speech Intensity in the Context of the MP Task (Objective 17)

The aim of this Objective was to examine the loudness perception ratings made by participants when completing the MP task in complete masking noise. To our knowledge, this is the first study to examine loudness perception ratings in complete masking noise. Since no auditory feedback was available during this task, the participants were to use any strategy they wanted to make their loudness ratings. If a participant inquired about how to rate their loudness, they were encouraged to use alternate methods such as ratings based on “how it feels” or “how much effort”.

Although participants accurately rated their loudness as successively louder with each MP condition, they were observed to have overall similar ratings of loudness whether in no noise or in complete masking noise. This did not align with the increased intensity that was produced in the complete masking noise condition and suggests that it is difficult to make loudness perception ratings when auditory feedback is completely blocked.

Interestingly, the PD and control group ratings were not statistically different, however a trend was observed in the data such that the control group rated their speech as louder in complete masking noise (consistent with the increase in intensity). The PD group was not observed to perceive an intensity increase when speaking in complete masking noise. This is in contrast to the PDs consistent overestimations of their loudness in all other conditions (no noise and in background noise). It is possible this is a reflection of a somatosensory deficit or deficit in sense of effort in addition to the auditory self-loudness deficit observed in the previous study objectives. The results related to the current objective indicate that complete masking noise differently affects controls and PDs and this difference is primarily related to an auditory feedback deficit.

4.9 Summary of Discussion

The current study contributes to our understanding of hypokinetic dysarthria in PD and advances our specific understanding of the role of auditory perception in PD-related hypophonia. Individuals with PD were observed to produce a flatter AIF response compared to the controls in all of the experiments in this study. The overlay of background noise, varying interlocutor distance, speech task, MP tasks, imitation tasks, and instructions to ignore auditory feedback had relatively little impact on this AIF response. These findings indicate the robustness of a reduced AIF response in PD and advance our understanding of a speech auditory-motor deficit in PD. Specifically, individuals with PD are suggested to either have abnormal perception of the intensity of their speech or were unable to appropriately integrate the auditory information of their speech for the production of intended intensity levels (auditory-motor goals).

The following 3 hypotheses are suggested based on preliminary evidence from the current study. We suggest that PDs may have 1) a primary deficit in the planning of internal intensity targets, 2) a deficit in the processing of external auditory feedback related to intensity, or 3) a deficit in the processes related to the integration of external self-loudness perception and internal estimation of self-loudness production. Results for tasks and conditions that have less reliance on external feedback and perhaps greater reliance on internal intensity planning were associated with the PD group having speech intensity that was closer to that of the control group (i.e. MP task). Conversely, tasks requiring more reliance on external feedback were associated with the PD group having speech intensity that was distinct from the control group (i.e. speech tasks such as conversation). Therefore, we propose that individuals with PD have a greater deficit in the processing of external auditory feedback (hypothesis 2) and in the integration of external and internal feedback processes (hypothesis 3). Although there may be a deficit in planning internal targets for speech production (hypothesis 1), or a reduced efference copy according to the DIVA model, this is less supported by the evidence in the current study. It is not completely rejected however, since the PD group was observed to show a deficit in gain setting for internally generated speech targets (i.e. MP task) such that the PD group produced a lower intensity of speech despite showing a successive increase in speech intensity across the MP conditions that was similar to the control group.

The current study provides new descriptions of the sensorimotor integration abnormalities in PD. Research that has examined sensorimotor integration as it relates to other motor movements have typically found “overreliance” of sensory information (such that in the absence of sensory input, individuals with PD have shown an increased deficit in motor production) and movement undershoot in the absence of this information (Almeida et al., 2005; Martens et al., 2013; Bronstein et al., 1990; Klockgether &

Dichgans, 1994; Rinalduzzi et al., 2015; Teulings et al., 2002). The current study showed movement undershoot in the form of reduced compensations to altered feedback, however responses in the absence of auditory feedback suggest the PD group has an "under reliance" on sensory information for speech motor movements. This under reliance is proposed because the PD group's speech was not affected when auditory feedback was blocked, suggesting the reliance or use of external feedback of their speech is reduced compared to the control group. This under reliance may be related to compensatory mechanisms of the PD motor speech system for the previously described deficit in processing externally generated feedback, the excessive inhibition of external feedback, or in the capacity to integrate sensory information.

It is possible that the sensorimotor integration abnormalities observed in PD are related to abnormal sensory gating. Sensory gating is the neurological process of filtering irrelevant or redundant sensory signals and the basal ganglia are thought to play a role in this process as it relates to motor function (Juri et al., 2011; Graybiel et al., 1994; Mink, 1996; Kaji, 2001). Previous studies suggest the possible role of abnormal sensory gating for PD motor movement (bradykinesia) (Conte et al., 2017) and describe potential task-related effects on the degree of sensory gating (grips tasks) (Lei et al., 2018). Gulberti and colleagues (2015) suggest reduced sensory gating in the auditory domain in PD (as evidenced from increased auditory evoked potentials to stimulus repetition; an indicator of lack of habituation to auditory stimulus presentation). How this relates to sensorimotor integration for speech is unclear. The reduced response by PD participants to altered auditory feedback in the current study suggests a reduced degree of sensory gating as it relates to auditory feedback for speech intensity production. It seems plausible that if the degree of sensory gating is reduced in PD, the amplitude of the intensity signal is increased, leading to abnormal responses to altered feedback, as well as overestimations of self-loudness. However this is speculative and future studies are recommended to examine auditory evoked potentials during altered intensity feedback and better understand sensory gating for PD-related hypophonia. Further recommendations include participants completing auditory-sensory tasks (e.g. loudness discrimination, just noticeable difference, loudness matching tasks, etc.) while speaking. In addition, studies could examine different speech tasks (e.g. conversation, vowel prolongation) as well as different speech measures (e.g. pitch, articulation) to determine whether task-based differences exist in sensory gating of speech in PD.

In the current study, a PD deficit related to the sensorimotor integration for speech intensity when speaking with clear communicative goals was observed and this was particularly pronounced when speaking in noisy environments. It is possible that individuals with PD have a pronounced difficulty maintaining communication goals in naturalistic environments due to the associated increase in demands, and this difficulty may be related to the observed deficits in processing and/or integrating of external feedback.

The current study supports and expands on previous literature related to abnormal loudness perception in PD specific to self-produced speech. In the current study, it is proposed that the overestimated self-loudness ratings are a result of a deficit in the external feedback system. In other words, the PD system may involve under reliance on external feedback, and we propose there may be an increased reliance (over-reliance) on internal predictions (feedforward processes), which leads to inaccuracy in loudness perceptions in this population. Mollaei and colleagues (2013) suggest that basal ganglia damage may be causing an amplification of reafferent sensory feedback (sensory information generated by self-produced movements). Similarly, Arnold and colleagues' findings (2014) indicate that in PD, there may not be adequate suppression of the auditory cortex while speaking. These findings may help to explain the inaccuracy in loudness perceptions observed in the current study.

4.10 Study Limitations and Directions for Future Research

The current study focused on average intensity across an utterance or condition, however it is possible that other speech parameters may have been affected by the AIF paradigm. Previous studies have suggested that changes in speech intensity are reflected in changes on other speech processes (e.g. articulation, vowel space, first and second formants) (Huber & Darling, 2011; Dromey & Ramig, 1998; Huber & Chandrasekaran, 2006), and so future studies are recommended to examine the impact of AIF on related speech parameters. Related to this, the method by which intensity adjustments were achieved by the different groups were not examined in this study. For example relative laryngeal or respiratory contributions and mouth opening dynamics may be investigated in future studies.

Variability across a task may be important to examine in future studies. The current study did not examine variation across utterances or the possible adaptation to AIF across and within a condition or task. It is possible that utterance length may play a role in this type of consideration with certain tasks such as conversation being more susceptible to possible adaptation effects. In addition, possible intensity declination effects are an important avenue for future research as Rosen and colleagues (2005) found task-based differences in this measure.

The PD participants in the current study were all selected based on the presence of hypophonia in their speech. Experimenter AA collected all data for this study and as an SLP graduate student with 4 years of experience with PD patients, assigned a rating of hypophonia severity (mild, moderate, severe, or a combination of two) based on subjective analysis of speech outside of the experimental protocol. Based on these perceptual ratings, on average the PD participants were rated as mild-moderate (range= mild – moderate/severe). In addition, objective measures of average speech intensity was compared between the PD and control groups and although on average the control group was louder than the PD group, no significant group differences were observed in any of the speech tasks (conversation, reading, vowel). This is consistent with the ratings of mild-moderate hypophonia. This presents a potential limitation of this study since it is unclear whether responses to AIF would be different in individuals with severe hypophonia. Despite the low hypophonia severity of the PD participants in the current study, the reduced responses to the AIF paradigm are robust, suggesting that the underlying deficit is present even with mild speech problems.

Another limitation of the current study is the heterogeneity in the presentation of the participants with PD in terms of the disease duration, severity of their symptoms and PD-related medication. The variability in disease severity presents a limitation of the current study, as do most studies of PD, as the range of motor and non-motor symptoms vary widely across the PD population (Chaudhuri, Healy, Schapira, 2006; Foltynie, Brayne, Barker, 2002).

The current protocol did not include testing the PD group in “on” versus “off” medication states. Therefore the role of the basal ganglia as it relates to PD auditory-motor dysfunction is unknown. Although the impact of dopaminergic medication on aspects of speech and auditory processing are unclear, future studies are recommended to examine the potential effects of medication on sensorimotor integration and specifically on the response to AIF.

Participant visits were scheduled so as to minimize possible fatigue, however no direct measures of fatigue were obtained for the current study. Fatigue can be a debilitating symptom in PD (Friedman, Abrantes, & Sweet, 2012). Fatigue has been associated with reduced communication participation (McAuliffe, Baylor, & Yorkston, 2017) and increased effort while speaking (Solomon & Robin, 2005). Therefore, future AIF studies should include measures of perceived fatigue. However a study by Makashay, Cannard, and Solomon (2015) indicated an overall fatigue-resistant speech system in PD speakers.

Speech-motor control involves the complex coordination of large groups of muscles across multiple systems, including phonatory, resonatory and respiratory systems. Sensory monitoring for speech intensity regulation may involve auditory processes as discussed in the current study, however it may also include other forms of sensory processing such as somatosensory and proprioception. It may be possible to alter or mask these other forms of sensory input in order to examine the relative contribution of each for speech intensity regulation in control as well as PD populations. The exclusion of these other processes is a potential limitation of the current study, however is suggested as an interesting avenue for future research.

4.11 Theoretical and Clinical Implications

The current study has important clinical and theoretical implications related to the understanding of auditory-motor speech processes in PD and also related to therapeutic considerations for PD-related hypophonia. Current treatment recommendations for PD-related hypophonia include training of internal targets (e.g. increased effort for speech loudness). Given the current proposed increased deficit in processing external feedback, integrating this information for motor production, and the deficit in self-loudness perception, (in comparison to a deficit in internal targets) then treatments that focus on enhancing or correcting auditory feedback deficits may need to be given greater consideration in future clinical investigations.

We suggest consideration of therapeutic options for re-training the processing and integrating of external feedback for speech intensity regulation. For example, visual feedback of speech intensity may be used to train auditory perception of external intensity stimuli as well as self-produced intensity targets. It may be possible to train the system over time, to rely on auditory information and use this information for appropriate speech intensity control.

There is also the potential for development of new training aids and assistive devices to provide accurate feedback related to speech intensity. For example, a speech intensity-monitoring device that could provide feedback signals (i.e. a warning tone) to the speaker when their speech intensity falls below a target loudness level. Future clinical investigations could explore the benefits of this type of external feedback monitoring device or use as a potential training system or as a long-term assistive device for improving hypophonia in PD.

Therapy options may also include external feedback-based communication training so that patients are better able to use communication-related cues (i.e. gestures, eye contact), and speaking conditions (i.e. interlocutor distance, background noise) to appropriately regulate their speech intensity.

4.12 Conclusion

The current study systematically manipulated auditory feedback in sensorimotor conditions that are known to modulate speech intensity in naturalistic contexts. Overall results indicate that individuals with PD display a reduced response to the altered intensity feedback in all speech tasks. These results significantly contribute to our understanding of sensorimotor integration in PD and suggest abnormal processing of auditory feedback for speech intensity regulation. There is preliminary evidence to indicate a specific deficit related to the processing and integration of external feedback for speech-motor production in PD, and this is distinguishable from controls. This work has important theoretical and clinical implications relative to our understanding of the role of auditory feedback for speech intensity control in PD populations.

References

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Gilmore, G., Delrobaei, M., & Jog, M. (2019). Effects of deep brain stimulation of the subthalamic nucleus settings on voice quality, intensity, and prosody in parkinson's disease: Preliminary evidence for speech optimization. *Canadian Journal of Neurological Sciences, 46(3),* 287-294

Abbruzzese, G., & Berardelli, A. (2003). Sensorimotor Integration in Movement Disorders. *Movement Disorders, 18(3),* 231–240.

Abur, D., Lester-Smith, R.A., Daliri, A., Lupiani, A.A., Guenther, F.H., Stepp, C.E. (2018). Sensorimotor adaptation of voice fundamental frequency in Parkinson’s disease. *Plos One, 13(1),* 1-21.

Adams, S.G. & Dykstra, A. (2009). *Hypokinetic dysarthria.* In M. R. McNeil (Ed.), Clinical Management of Sensorimotor Speech Disorders. New York: Thieme Pub.

Adams, S.G., Dykstra, A., Abrams, K., Winnell, J., Jenkins, M., & Jog, M. (2006). Conversational speech intensity under different noise conditions in hypophonia and Parkinson's. *Canadian Acoustics, 34,* 96-97.

Adams, S.G., Dykstra, A. & Jog, M. (2012). A Comparison of Throat and Head Microphones in a PDA-based Evaluation of Hypophonia in Parkinson’s Disease.

Journal of Medical Speech Language Pathology, 20(4), 1-6.

Adams, S.G., Haralabous, O., Dykstra, A., Abrams, K., & Jog, M. (2005). Effects of multi-talker background noise on the intensity of spoken sentences in Parkinson's disease. *Canadian Acoustics, 33,* 94-95.

Adams, S.G. and Lang, A.E. (1992). Can the Lombard effect be used to improve low voice intensity in Parkinsonian speakers. *European Journal of Disorders of Communication, 27(2),* 121-127.

Adams, S.G., Moon, B., Dykstra, A., Abrams, K., Jenkins, M. & Jog, M. (2006). Effects of multi- talker noise on conversational speech in Parkinson's disease. *Journal of Medical Speech-Language Pathology, 14,* 221-228.

Adams, S.G., Winnell, J., & Jog, M. (2010). Effects of interlocutor distance: multi-talker background noise, and a concurrent manual task on speech intensity in Parkinson's disease. *Journal of Medical Speech-Language Pathology, 18(4),* 1–8.

Almeida, Q.J., Frank, J.S., Roy, E.A., Jenkins, M.E., Spaulding, S., Patla, A.E., & Jog, M.S. (2005). An evaluation of sensorimotor integration during locomotion toward a target in Parkinson's disease. *Neuroscience 134:* 283–293.

Alexander, G., & Crutcher, M. (1990). Functional architecture of basal ganglia circuits:

Neural substrates of parallel processing. *Trends in Neurosciences 13(7),* 266–271.

American National Standards Institute. (1973). *American national psychoacoustical terminology, S3.20,* American Standards Association.

Arnold, C., Gehrig, J., Gispert, S., Seifried, C., & Kell, C. A. (2014). Clinical Pathomechanisms and Compensatory Efforts Related to Parkinsonian Speech. *NeuroImag, 4,* 82–97.

Baker, E., Blumstein, S., & Goodglass, H. (1981). Interaction between phonological and semantic factors in auditory comprehension. *Neuropsychologia 19(1),* 1–15.

Balleine, B., Delgado, M., Hikosaka, O. (2007). The role of the dorsal striatum in reward and decision-making. *Journal of Neuroscience, 27(31),* 8161-8165.

Bauer, J.J., Mittal, J., Larson, C.R., & Hain, T.C. (2006). Vocal responses to unanticipated perturbations in voice loudness feedback: An automatic mechanism for stabilizing voice amplitude. *Journal of Acoustical Society of America, 119 (4),* 2363-2371.

Baum, S., & Pell, M. (1999). The neural bases of prosody: insights from lesion studies and neuroimaging. *Aphasiology 13,* 581–608.

Bays, P.M., Wolpert, D.M., Flanagan, J.R. (2005). Perception of the consequences of self-action is temporally tuned and event driven. *Current Biology, 15*, 1125–1128.

Behroozmand, R., Karvelis, L., Liu, H., & Larson. C. (2009). Vocalization-induced enhancement of the auditory cortex responsiveness during voice F0 feedback perturbation. *Clinical Neurophysiology 120(7),* 1303–1312.

Black, J.W. (1951). The effect of noise induced temporary deafness upon vocal intensity. *Speech Monographs, 18,* 74-77.

Boersma, P., & Weenink, D. (2011). Praat: Doing phonetics by computer [Computer program]. Version 5.2.26. Retrieved from: http://www.praat.org/

Boller, F., Passafiume, D., Keefe, N.C., Rogers, K., Morrow, L., Kim, Y. (1984). Visuospatial impairment in Parkinson's disease. Role of perceptual and motor factors. *Archives of Neurology, 41 (5),* 485-90.

Bora, E., Walterfang, M., & Velakoulis, D. (2015). Theory of mind in Parkinson's disease: A meta-analysis. *Behavioural brain research, 292*, 515–20.

Braak, H., Ghebremedhin, E., Rüb, U., Bratzke, H., & Del Tredici, K. (2004). Stages in the development of Parkinson's disease-related pathology. *Cell and tissue research, 318*(1), 121–34.

Brajot, F., Shiller, D.M., Gracco, V.L. (2016). Autophonic loudness perception in Parkinson’s disease. *Journal of Acoustical Society of America, 139(3),* 1364-71.

Brin, M. F., Blitzer, A., Fahn, S., & Lovelace, R. E. (1989). Adductor Laryngeal Dystonia (Spastic Dysphonia): Treatment with Local Injections of Botulinum Toxin (Botox). *Movement Disorders, 4(4),* 287–296.

Bronstein, A. M., Hood, J. D., & Gresty, M. A. (1990). Visual Control of Balance in Cerebellar and Parkinsonian Syndromes. *Brain, 113,* 767–779.

Brown, A., & Docherty, G.J. (1995). Phonetic variation in dysarthric speech as a function of sampling task. *European Journal of Disorders of Communication, 30,* 17–35.

Brug, M. P. V. D., Singleton, A., Gasser, T., & Lewis, P. A. (2015). Parkinson’s disease : From human genetics to clinical trials. *Science Translational Medicine, 8(305).*

Buchsbaum, B., Hickok, G., & Humphries, C. (2001). Role of left posterior superior temporal gyrus in phonological processing for speech perception and production. *Cognitive Science 25(5),* 663–678.

Burnett, T.A., Freedland, M.B., Larson, C.R., Hain, T.C. (1998). Voice F0 Responses to Manipulations in Pitch Feedback. *Journal of Acoustical Society of America, 103,* 3153-3161.

Callan, D., Jones, J., Callan, A., & Akahane-Yamada, R. (2004). Phonetic perceptual identification by native and second language speakers differentially activates brain regions involved with acoustic phonetic processing and those involved with articulatory auditory/orosensory internal models. *NeuroImage 22(3),* 1182–1194.

Chang, E.F., Niziolek, C.A., Knight, R.T., Nagarajan, S.S., & Houde. J.F. (2013). Human cortical sensorimotor network underlying feedback control of vocal pitch. *Proceedings of the National Academy of Sciences of the United States of America, 110(7),* 2653-8.

Chen, X., Zhu, X., Wang, E.Q., Chen, L., Li, W., Chen, Z., & Liu, H. (2013). Sensorimotor control of vocal pitch production in Parkinson's disease. *Brain Research, 1527,* 99–107.

Cheyne, H.A., Kalgaonkar, K., Clements, M., & Zurek, P. (2009). Talker-to-listener distance effects on speech production and perception. *Journal of Acoustical Society of America, 126 (4),* 2052-2060.

Chaudhuri, K.R., Healy, D.G., Schapira, A.H. (2006). Non-motor symptoms of Parkinson’s disease: diagnosis and management. *The Lancet Neurology. 5(3),* 235, 45.

Christoffels, I., Formisano, E., & Schiller, N. (2007). Neural correlates of verbal feedback processing: An fMRI study employing overt speech. *Human Brain Mapping 28(9),* 868–879.

Clark, J. P., Adams, S. G., Dykstra, A. D., & Moodie, S. (2014). Journal of Communication Disorders Loudness perception and speech intensity control in Parkinson' s disease, *51*, 1–12.

Clark, J. (2012). Loudness perception and speech intensity control in Parkinson's disease. Unpublished master's thesis. University of Western Ontario, London, Ontario, Canada.

Conte, A., Khan, N., Defazio, G., Rothwell, J. C., & Berardelli, A. (2013). Pathophysiology of somatosensory abnormalities in Parkinson disease. *Nature reviews. Neurology*, *9*(12), 687–97.

Conte, A., Belvisi, D., Tartaglia, M., Cortese, F.N., Baione, V., Battista, E., …Berardelli, A. (2017). Abnormal temporal coupling of tactail perception and motor action in Parkinson's disease. *Frontiers in Neurology, 8,* 1-9.

Coutinho, S. B., Oliveira, G., & Behlau, M. (2009). Voice and speech of individuals with Parkinson´s Disease during amplification, delay and masking situations. *Pró-Fono Revista de Atualização Científica, 21(3),* 219–224.

Cunnington, R., Bradshaw, J.L., Iansek, R., (1996). The role of the supplementary motor area in the control of voluntary movement. *Human Movement Science, 15,* 627–647.

Curio, G., Neuloh, G., Numminen, J., Jousmaki, V., & Hari, R. (2000). Speaking modifies voice evoked activity in the human auditory cortex. *Human Brain Mapping 9(4)*, 183–191.

Daniel, R., Pollmann, S. (2014). A universal role of the ventral striatum in reward-based learning: Evidence from human studies. *Neurobiol Learn Mem*, 90-100.

Darley, F.L., Aronson, A.E., & Brown, J.R. (1969). Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research, 12,* 246–269.

DeKeyser, K., Santens, P., Bockstael, A., Botteldooren, D., Talsma, D., De Vos, S., Van Cauwenberghe, M., Verheugen, F., Corthals, P., & De Letter, M. (2016). The Relationship Between Speech Production and Speech Perception Deficits in Parkinson's Disease. *Journal of Speech, Language and Hearing Research*, 1-17.

Dorsey, E. R., Constantinescu, R., Thompson, J. P., Biglan, K. M., Holloway, R. G., Kieburtz, K., Marshall, F. J., et al. (2007). Projected number of people with Parkinson disease in the most populous nations, 2005 through 2030, *Neurology, 8,* 2005–2007.

Dromey, C. & Adams, S.G. (2000). Loudness perception and hypophonia in Parkinson's disease, *Journal of Medical Speech-Language Pathology, 8, 255-259*.

Dromey, C., & Ramig, L.O. (1998). Intentional changes in sound pressure level and rate: their impact on measures of respiration, phonation, and articulation. *Journal of Speech, Language and Hearing Research, 41,* 1003–1018.

Dykstra, A. D., Adams, S. G., & Jog, M. (2015). Examining the relationship between speech intensity and self-rated communicative effectiveness in individuals with Parkinson's disease and hypophonia. *Journal of Communication Disorders, 56,* 103–112.

Dykstra, A. D., Hakel, M. E., & Adams, S. G. (2007). Application of the ICF in Reduced Speech Intelligibility in Dysarthria, *Seminars in Speech Language, 28(4*), 301–311.

Duffy, J.R. (2013). Motor Speech Disorders: Substrates, Differential Diagnosis, and Management. Missouri: Elsevier Health Sciences.

Eliades, S., & Wang, X. (2008). Neural substrates of vocalization feedback monitoring in primate auditory cortex. *Nature 453(7198),* 1102–1106.

Fairbanks, G. (1954). Systematic research in experimental phonetics: A theory of speech mechanism as a servosystem. *Journal of Speech and Hearing Disorders, 19*, 133–139.

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behavioral Research Methods, 39(2), 175-191.

Foltynie, T., Brayne, C., Barker, R.A. (2002). The heterogeneity of idiopathic Parkinson’s disease. *Journal of Neurology, 249(2),* 138–45.

Fox, C. & Ramig, L. (1997). Vocal sound pressure level and self-perception of speech and voice in men and women with idiopathic Parkinson disease. *American Journal of Speech-Language Pathology, 6,* 85–94.

Friederici, A. D., & Alter, K. (2004). Lateralization of auditory language functions: A dynamic dual pathway model. *Brain and Language, 89,* 267–276.

Friedman, J., Abrantes, A., Sweet, L. (2011). Fatigue in Parkinson’s disease. *Expert Opinion on Pharmacotherapy, 12(13),* 1999-2007.

Fu, C., Vythelingum, G., Brammer, M., Williams, S., Amaro, E., Andrew, C., Yaguez, L., van Haren, N., Matsumoto, K., McGuire, P. (2006). An fMRI study of verbal self-monitoring: Neural correlates of auditory verbal feedback. *Cerebral Cortex 16(7).*

969–977.

Garnier, M., Henrich, N., & Dubois, D. (2010). Influence of Sound Immersion and Communicative Interaction on the Lombard Effect. *Journal of Speech, Language, and Hearing Research*, *53,* 588-608.

Gawel, M.J., Das, P., Vincent, S., Rose, F.C. (1981). Visual and auditory evoked responses in patients with Parkinson’s disease. Journal of Neurology, *Neurosurgery & Psychiatry, 44(3)*, 227–232.

Glickstein,M. (1997). Mossy fibre sensory input to the cerebellum. *Progress in Brain Research 114*, 251–259.

Goetz, C. G., & Pal, G. (2014). Initial management of Parkinson’s disease. *British Medical Journal, 349.*

Golfinopoulos, E., Tourville, J. A., & Guenther, F. H. (2010). The integration of large-scale neural network modeling and func- tional brain imaging in speech motor control. *NeuroImage, 52,* 862–874.

Govil, N., Akinin, A., Ward, S., Snider, J., Plank, M., Cauwenberghs, G., & Poizner, H. (2013). The Role of Proprioceptive Feedback in Parkinsonian Resting Tremor. *Conference Proceedings IEEE Eng Medical Biological Society,* 4969–4972.

Graybiel, A.M., Aosaki, T., Flaherty, A.W., Kimura, M. (1994). The basal ganglia and adaptive motor control. *Science, 265,* 1826–31.

Greenlee, J., Jackson, A., Chen, F., Larson, C., Oya, H., Kawasaki, H., Chen, H., & Howard III, M. (2011). Human auditory cortical activation during self vocalization. *PLoS ONE 6(3),* e14744.

Guenther, F. H. (1994). A neural network model of speech acquisition and motor equivalent speech production. *Biological Cybernetics, 72*, 43-53.

Gulberti, A., Hamel, W., Buhmann, C., Boelmans, K., Zittel, S., Gerloff, C.,…Moll, C.K.E. (2015). Subthalamic deep brain stimulation improves auditory sensory gating deficit in Parkinson's disease. *Clinical Neurophysiology, 126,* 565-574.

Hammer, M. J., & Barlow, S. M. (2010). Laryngeal somatosensory deficits in Parkinson's disease: implications for speech respiratory and phonatory control. *Experimental brain research, 201*(3), 401–9.

Hansen, H.L., & Boril, H. (2018). On the issues of intra-speaker variability and realism in speech, speaker, and language recognition tasks. *Speech Communication, 101*, 94-108.

Haslinger, B., Erhard, P., Kampfe, N., Boecker, H., Rummeny, E., Schwaiger, M., Conrad, B., Ceballos-Baumann, A.O. (2001). Event-related functional magnetic resonance imaging in Parkinson's disease before and after levodopa. *Brain, 124,* 558–570.

Hegland, K. W., Troche, M., & Brandimore, A. (2019). Relationship between respiratory sensory perception, speech, and swallow in parkinson's disease. *Movement Disorders Clinical Practice, 6(3),* 243-249.

Heinks-Maldonado, T. H., & Houde, J. F. (2005). Compensatory responses to brief perturbations of speech amplitude, *ARLO, 6 (3),* 131-37.

Hickok, G., Buchsbaum, B., Humphries, C., & Muftuler, T. (2003). Auditory-motor interaction revealed by fMRI: Speech, music and working memory in area Spt. *Journal of Cognitive Neuroscience 15(5),* 673–682.

Hickok, G., & Poeppel, D. (2000). Towards a functional neuro- anatomy of speech perception. *Trends in Cognitive Sciences, 4,* 131–138.

Ho, A. K., Bradshaw, J. L., & Iansek, R. (2000). Volume Perception in Parkinsonian Speech. *Movement Disorders, 15(6),* 1125–1131.

Ho, A. K., Bradshaw, J. L., Iansek, R., & Alfredson, R. (1999). Speech volume regulation in Parkinson’s disease : effects of implicit cues and explicit instructions, *Neuropsychologia, 37*, 1453–1460.

Hobson, D.E. (2003). Clinical Manifestations of Parkinson's disease and Parkinsonism. *Canadian Journal of Neurological Sciences, 30,* S2-S9.

Houde, J. & Jordan, M. (2002). Sensorimotor adaptation of speech I: Compensation and adaptation. *Journal of Speech, Language, and Hearing Research 45(2)*, 295–310.

Huang, X., Chen, X., Yan, N., Jones, J., Wang, E., Chen, L., Guo, Z., Li, W., Liu, P., Liu, H. (2016). The Impact of Parkinson’s Disease on the Cortical Mechanisms That Support Auditory–Motor Integration for Voice Control. *Human Brain Mapping 37,* 4248–4261.

Huber, J. E., & Chandrasekaran, B. (2006). Effects of increasing sound pressure level on lip and jaw movement parameters and consistency in young adults. *Journal of Speech, Language, and Hearing Research, 49(6),* 1368-79.

Huber, J. E., & Darling, M. (2012). Effect of Parkinson Disease on the Production of Structured and Unstructured Speaking Tasks: Respiratory physiologic and linguistic considerations. *Journal of Speech, Language, and Hearing Research, 54(1),* 33–46.

Iulianella, I., Adams, S. G., & Gow, A. K. (2008). Effects of sub-thalamic deep brain stimulation on speech production in parkinson's Disease: A Critical Review of the Literature. *Canadian Journal of Speech-Language Pathology and Audiology, 32,* 85–91.

Jankovic, J. (2008). Parkinson’s disease: clinical features and diagnosis. *Journal of neurology, neurosurgery, and psychiatry*, *79*(4), 368–76.

Junqua, J. C., Finckle, S., & Field, K. (1999). The Lombard effect: A reflex to better communicate with others in noise. *In Proceedings of ICASSP ’99, the International Conference on Acoustics, Speech and Signal Processing,* 2083–2086.

Jurgens, U. (2002). Neural pathways underlying vocal control. Neuroscience *Biobehavioral Reviews 26(2),* 235–258.

Juri, C., Rodriguez-Oroz, M., Obeso, J.A. (2011). Functional organization of the basal ganglia: dopaminergic and non-dopaminergic features. In: C.W. Olanow, F. Stocchi, & A.E. Lang (Eds.), *Parkinson’s disease: non-motor and non-dopaminergic features.* Chichester: Wiley-Blackwell.

Kaji, R. (2001). Basal ganglia as a sensory gating devise for motor control. *The Journal of Medical Investigation, 48,* 142-145.

Kaji, R., Urushihara, R., Murase, N., Shimazu, H., Goto, S. (2005). Abnormal sensory gating in basal ganglia disorders. *Journal of Neurology, 252 (4),* IV13-16.

Kennedy, L. (1930). *Studies in the Speech of the Feeble-Minded.* Wisconsin: University of Wisconsin-Madison.

Kent, R. D. (2004). The uniqueness of speech among motor systems. *Clinical Linguistics & Phonetics, 18,* 495–505.

Kent, R.D., Kent, J.F., Rosenbek, J.C., Vorperian, H.K., Weismer, G. (1997). A speaking task analysis of the dysarthria in cerebellar disease. *Folia Phoniatrica et Logopaedica*, *49*, 63–82.

Kern, D. S., & Kumar, R. (2007). Deep brain stimulation. *The neurologist*, *13*(5), 237–52.

Klockgether T., Dichgans J. (1994). Visual control of arm movement in Parkinson's disease. *Movement Disorders, 9,* 48–56.

Knowles, T., Adams, S., Abeyesekera, A., Mancinelli, C., Gilmore, G., & Jog, M. (2018). Deep brain stimulation of the subthalamic nucleus parameter optimization for vowel acoustics and speech intelligibility in Parkinson's disease. *Journal of Speech, Language, and Hearing Research, 61(3),* 510-524.

Kwan, L. C., & Whitehill, T. L. (2011). Perception of speech by individuals with

Parkinson’s disease: a review. *Parkinson’s disease, 2011*, 389767.

Lai, S.W., Liao, K.F., Lin, C.L., Lin, C.C., & Sung, F.C. (2014). Hearing loss may be a non-motor feature of Parkinson’s disease in older people in Taiwan. *European journal of neurology : the official journal of the European Federation of Neurological Societies*, *21*(5), 752–7.

Lametti, D.R., Nasir, S.M., Ostry, D.J. (2012). Sensory preference in speech production revealed by simultaneous alteration of auditory and somatosensory feedback. *Journal of Neuroscience, 32,* 9351–9358.

Lane H., Denny M., Guenther F. H., Matthies M. L., Ménard L., Perkell J. S., Stockmann, E., Tiede, M., Vic, J., Zandipour M. (2005). Effects of bite blocks and hearing status on vowel production. *The Journal of the Acoustical Society of America, 118,* 1636–1646.

Lane, H., Catania, A., & Stevens, S. (1961). Voice Level: Autophonic Scale, Perceived Loudness and Effects of Sidetone. *Journal of the Acoustic Society of America, 33,* 160-167.

Lane, H., & Tranel, B. (1971). The Lombard sign and the role of hearing in speech. *Journal of Speech and Hearing Research, 14,* 677-709.

Lane, H., Tranel, B., & Sisson, C. (1969). Regulation of Voice Communication by Sensory Dynamics. *Journal of the Acoustic Society of America*, *47*,618-623.

Langston, J. W., Forno, L. S., Rebert, C. S., & Irwin, I. A. N. (1984). Selective nigral toxicity after systemic administration of 1-methyl-4-phenyl-1 , 2 , 5 , 6-tetrahydropyrine (MPTP) in the squirrel monkey. *Brain Research, 292*, 390–394.

Larson, C., Altman, K., Liu, H., & Hain, T. (2008). Interactions between auditory and somatosensory feedback for voice F0 control. *Experimental Brain Research, 187(4),* 613-21).

Lei, Y., Ozdemir, R.A., Perez, M.A. (2018). Gating of sensory input at subcortical and cortical levels during grasping in humans. *The Journal of Neuroscience, 38 (33),* 7237-7247.

Lindblom, B. (1990). Explaining phonetic variation: A sketch of the H&H theory. In W. J. Hardcastle & A. Marchal (Eds.), Speech production and speech modeling (pp. 403–439). Dordrecht, The Netherlands: Kluwer Academic.

Liu, H., Wang, E.Q., Metman, L.V., & Larson, C.R. (2012). Vocal Responses to Perturbations in Voice Auditory Feedback in Individuals with Parkinson's Disease. *PLoS ONE, 7(3),* e33629.

Logemann, J.A., Fisher, H.B., Boshes, B., et al. (1978). Frequency and co- occurrence of vocal tract dysfunctions in the speech of a large sample of Parkinson patients. *Journal of Speech and Hearing Disorders, 43,* 47–57.

Lombard, É. (1911). Le signe de l'élévation de la voix. *Annales des Maladies de l'Oreille et du Larynx. XXXVII (2),* 101–9.

Luo, J., Hage, S.R., & Moss, C.F. (2018). The Lombard Effect: From Acoustics to Neural Mechanisms. *Trends in Neuroscience, 41(12),* 938-949.

Lukhanina, E. P., Kapustina, M. T., Berezetskaya, N. M., & Karaban, I. N. (2009). Reduction of the postexcitatory cortical inhibition upon paired-click auditory stimulation in patients with Parkinson’s disease. *Clinical Neurophysiology, 120,* 1852–1858.

Ma, L., Chan, P., Gu, Z., Li, F., & Feng, T. (2015). Heterogeneity among patients with Parkinson ’ s disease : Cluster analysis and genetic association. *Journal of the Neurological Sciences, 351,* 41–45.

Makashay, M.J., Cannard, K.R., Solomon, N.P. (2015). Speech-related fatigue and fatigability in Parkinson's disease. *Clinical Linguistics & Phonetics, 29(1),* 27-45.

Martens, K.A., Ellard, C.G., & Almeida, Q.J. (2013). Dopaminergic contributions to distance estimation in Parkinson's disease: A sensory-perceptual deficit? *Neuropsychologia, 51,* 1426-1434.

McAuliffe, M. J., Baylor, C.R., Yorkston, K.M. (2017). Variables associated with communicative participation in Parkinson's disease and its relationship to measures of health-related quality-of-life. *International Journal of Speech Language Pathology, 19(4),* 407-417.

McCaig, C. M., Adams, S. G., Dykstra, A. D., & Jog, M. (2015). Effect of concurrent walking and interlocutor distance on conversational speech intensity and rate in Parkinson’s disease. *Gait & posture, 43,* 132–136.

Meissner, I., Sapir, S., Kokmen, E., Stein, S. D., & Report, C. (1987). The Paramedian Diencephalic Syndrome: A Dynamic Phenomenon, *Stroke, 18(2),* 380–386.

Miceli, G., Gainotti, G., Caltagirone, C., & Masullo. C. (1980). Some aspects of phonological impairment in aphasia. *Brain and Language 11(1),* 159–169.

Miller, N., Noble, E., Jones, D., & Burn, D. (2006). Life with communication changes in Parkinson's disease. *Age and Ageing, 35(3),* 235-9.

Mink, J.W. (1996). The basal ganglia: focused selection and inhibition of competing motor programs. *Progress in Neurobiology, 50,* 381–425.

Mollaei, F., Shiller, D.M., Gracco, V.L. (2013). Sensorimotor adaptation of speech in Parkinson's disease. *Movement Disorders, 28,* 1668-1674.

Mollaei, F., Shiller, D., Baum, S., Gracco, V. (2016). Sensorimotor control of vocal pitch and formant frequencies in Parkinson's disease. *Brain Research, 1646,* 269-277.

Moon, B. (2005). Effect of background noise, listener context and speech tasks and requests for clarification on speech intensity in Parkinson's disease. Unpublished master's thesis. University of Western Ontario, London, Ontario, Canada.

Munhall, K.G., MacDonald, E.N., Byrne, S.K., & Johnsrude, I. (2009). Talkers alter vowel production in response to real-time formant perturbation even when instructed not to compensate. *Journal of the Acoustical Society of America, 125(10)*, 384-90.

Murofushi, T., Yamane, M., & Osanai, R. (1992). Stapedial reflex in Parkinson's disease. *ORL, 54(5),* 255–258.

Neel, A. T. (2009). Effects of Loud and Amplified Speech on Sentence and Word Intelligibility in Parkinson Disease. *Journal of Speech, Language, and Hearing Research, 52(4),* 1021–1033.

New, A., Robin, D., Parkinson, A., Eickhoff, C., Reetz, K., Hoffstaedter, F., Mathys, C., Sudmeyer, M., Grefkes, C., Larson, C., Ramig, L., Fox, P., Eickhoff, S. (2015). The intrinsic resting state voice network in Parkinson's disease. *Human Brain Mapping, 36(5)*, 1951-62.

Nixon, P.D. & Passingham, R.E. (1998). The striatum and self-paced movements. *Behavioral Neuroscience, 112,* 719–724.

Olanow, C. W., & Tatton, W. G. (1999). Etiology and Pathogenesis of Parkinson's Disease. *Annual Review of Neuroscience, 22,* 123–44.

Parkinson, A., Flagmeier, S., Manes, J., Larson, C., Rogers, B., & Robin, D. (2012). Understanding the neural mechanisms involved in sensory control of voice production. *NeuroImage 61(1),* 314–322.

Parkinson Society Canada. (2003). *Parkinson's disease: Social and Economic Impact* (Publication No. 0-9733421-0-2). Health Canada.

Patel, N., Jankovic, J., & Hallett, M. (2014). Sensory aspects of movement disorders. *Lancet Neurol, 13, 1*00-12.

Patel, S., Nishimura, C., Lodhavia, A., Korzyukov, O., Parkinson, A., Robin, D.A., Larson, C.R. (2014). Understanding the mechanisms underlying voluntary responses to pitch-shifted auditory feedback. *Journal of Acoustical Society of America, 135(5),* 3036-44.

Patel, R., & Schell, K.W. (2008). The influence of linguistic content on the Lombard effect. *Journal of Speech, Language, and Hearing Research, 51(1),* 209-20.

Perkell, J. S., Denny, M., Lane, H., Guenther, F. H., Matthies, M. L., Tiede, M., Vick, J., Zandipour, M., and Burton, E. (2007). Effects of masking noise on vowel and sibilant contrasts in normal-hearing speakers and post-lingually deafened cochlear implant users. *J. Acoust. Soc. Am. 121,* 505– 514.

Pick, H.L., Siegel, G.M., Fox, P.W., Garber, S.R., Kearney, J.K. (1989) Inhibiting the Lombard effect. *Journal of Acoustical Society of America, 85,* 894–900.

Pulvermuller, F. (2005). Brain mechanisms linking language and action. *Nature Reviews Neuroscience, 6,* 576–582.

Purcell, D.W., & Munhall, K.G. (2006). Adaptive control of vowel formant frequency: evidence from real-time formant manipulation. *Journal of the Acoustical Society of America, 120,* 966–977.

Ramig, L. O., Countryman, S., O'Brien, C., Hoehn, M., & Thompson, L. (1996). Intensive speech treatment for patients with parkinson's disease: Short- and long term comparison of two techniques. *Neurology, 47(6),* 1496-1504.

Ramig, L. O., & Dromey, C. (1996). Aerodynamic mechanisms underlying treatment related changes in vocal intensity in patients with parkinson disease. *Journal of Speech, Language, and Hearing Research, 39(4),* 798-807

Rektorova, I., Mikl, M., Barrett, J., Marecek, R., Rektor, I., Paus, T. (2012). Functional neuroanatomy of vocalization in patients with Parkinson's disease. *Journal of Neurological Science, 313(1-2),* 7-12.

Rinalduzzi, S., Trompetto, C., Marinelli, L., Alibardi, A., Missori, P., Fattapposta, F., Pierelli, F., & Currà, A. (2015). Balance Dysfunction in Parkinson's Disease, *BioMed Research International, 2015*.

Rosen, K.M., Kent, R.D., & Duffy, J.R. (2005). Task-based profile of vocal intensity decline in Parkinson's disease. *Folia Phoniatrica et Logopaedica, 57,* 28–37.

Rosen, K. M., Kent, R. D., Delaney, A. L., & Duffy, J. R. (2006). Parametric quantitative acoustic analysis of conversation produced by speakers with dysarthria and healthy speakers. *Journal of Speech, Language, and Hearing Research, 49,* 395–411.

Sapir, S. (2014). Multiple factors are involved in the dysarthria associated with Parkinson's disease: a review with implications for clinical practice and research. *Journal of Speech, Language & Hearing Research, 57,* 1330-1343.

Schneider, J. S., Diamond, S. G., & Markham, C. H. (1986). Deficits in Orofxial Sensorimotor Function in Parkinson’s Disease. *Annals of Neurology, 19(3),* 275–282.

Shadmehr, R., Smith, M. A., and Krakauer, J. W. (2010). Error correction, sensory prediction, and adaptation in motor control. *Ann. Rev. Neurosci. 33,* 89–108.

Siegel, G.M. & Pick, H.L. (1974). Auditory feedback in the regulation of voice. *Journal of the Acoustical Society of America, 56(5),* 1618-24.

Sober, S. & Sabes, P. (2005). Flexible strategies for sensory integration during motor planning. *Nature Neuroscience, 8(4),* 490-7.

Solomon, N.P. & Robin, D.A. (2005). Perceptions of effort during handgrip and tongue elevation in Parkinson's disease. *Parkinsonism & Related Disorders, 11(6),* 353-61.

Van Summers, W., Pisoni, B., Bernacki, H., Pedlow, R., & Stokes, M. (1988). Effect of noise on speech production: Acoustic and perceptual analyses. *The Journal of the Acoustical Society of America, 84,* 917–928.

Tatton, W.G., Eastman, M.J., Bedingham, W., Verrier, M.C., Bruce, I.C. (1984). Defective utilization of sensory input as the basis for bradykinesia, rigidity and decreased movement repertoire in Parkinson's disease: a hypothesis. *Canadian Journal of Neurological Science,* 136-43.

Teulings, H. L., Contreras-Vidal, J. L., Stelmach, G. E., & Adler, C. H. (2002). Adaptation of handwriting size Underdistorted visual feedback in patients with Parkinson’s disease and elderly and young controls. *Journal of Neurology, Neurosurgery, and Psychiatry, 72(3),* 315–324.

Tourville, J.A., Reilly, K.J., Guenther, F.H. (2008) Neural mechanisms underlying auditory feedback control of speech. *NeuroImage, 39,* 1429–1443.

Toyomura, A., Koyama, S., Miyamaoto, T., Terao, A., Omori, T., Murohashi, H., Kuriki, S. (2007). Neural correlates of auditory feedback control in human. *Neuroscience 146(2),* 499–503.

Traunmüller, H. & Eriksson, A. (2000). Acoustic effects of variation in vocal effort by men, women, and children. *Journal of Acoustical Society of America, 107,* 3438–3451.

van de Ven, V., Esposito, F., & Christoffels. I. (2009). Neural network of speech monitoring overlaps with overt speech production and comprehension networks: A sequential spatial and temporal ICA study. *NeuroImage 47(4)*, 1982–1991.

Vinney, L. A., van Mersbergen, M., Connor, N. P., & Turkstra, L. S. (2016). Vocal control: Is it susceptible to the negative effects of self-regulatory depletion? *Journal* *of Voice, 30(5),* 638.e21-638.e31

Vitale, C., Marcelli, V., Allocca, R., Santangelo, G., Riccardi, P., Erro, R., Amboni, M., et al. (2012). Hearing impairment in Parkinson’s disease: expanding the nonmotor phenotype. *Movement disorders : official journal of the Movement Disorder Society*, *27*(12), 1530–5.

Voss, M., Bays, P.M., Rothwell, J.C., Wolpert, D.M. (2007). An improvement in perception of self-generated tactile stimuli following theta-burst stimulation of primary motor cortex. *Neuropsychologia, 45,* 2712–2717.

Waldstein, R. S. (1990). Effects of postlingual deafness on speech production: Implications for the role of auditory feedback. *The Journal of the Acoustical Society of America, 88,* 2099–2114.

Warren, R. M. (1973). Quantification of Loudness. *The American Journal of Pscyhology, 86(4),* 807-825.

Wertheimer, J., Gottuso, A. Y., Nuno, M., Walton, C., Duboille, A., Tuchman, M., & Ramig, L. (2014). The impact of STN deep brain stimulation on speech in individuals with Parkinson’s disease : The patient's perspective. *Parkinsonism and Related Disorders, 20*, 16–21.

Wiener, N. (1948). *Cybernetics: Control and Communication in the Animal and the Machine.* New York: John Wiley & Sons, Inc.

Winkworth, A.L., Davis, P.J, Ellis, E., Adams, R.D. (1994). Variability and consistency in speech breathing during reading: Lung volumes, speech intensity, and linguistic factors. *Journal of Speech and Hearing Research, 37,* 535–556.

Wolpert, D.M. & Ghahramani, Z. (2000). Computational principles of movement neuroscience. *Nature Neuroscience, 3*, 1212–7.

Yeterian, E., & Pandya, D. (1998). Corticostriatal connections of the superior temporal region in rhesus monkeys. *Journal of Comparative Neurology 399(3),* 384–402.

Yorkston, K.M., Beukelman, D.R., & Tice, R. (1996). Sentence intelligibility test. Lincoln, NE: Institute for Rehabilitation Science and Engineering at Madonna Rehabilitation Hospital.

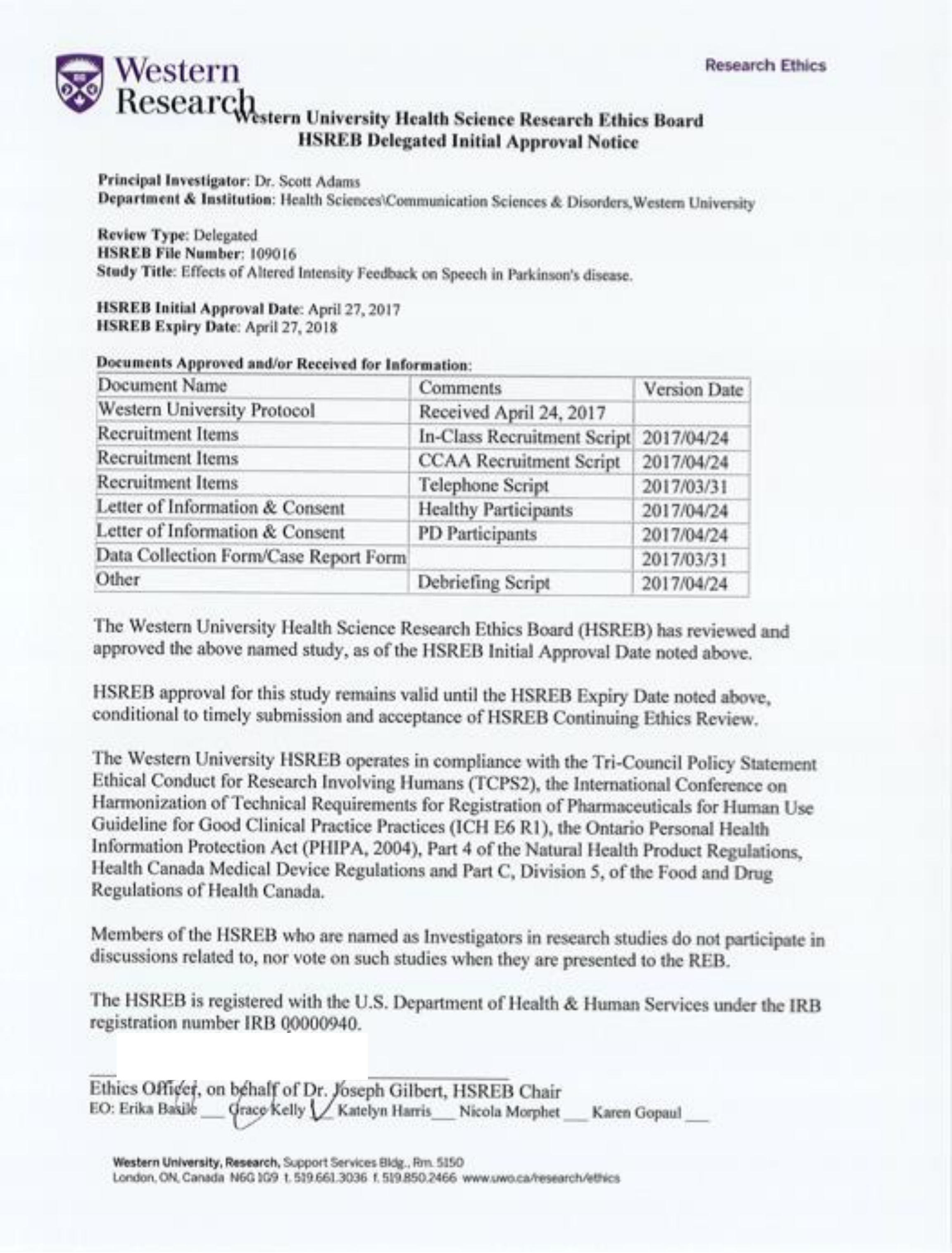
Yýlmaz, S., Karalý, E., Tokmak, A., Güçlü, E., Koçer, A., & Oztürk, O. (2009). Auditory evaluation in Parkinsonian patients. *European archives of otorhinolaryngology, 266*(5), 669–71.

Zarate, J., & Zatorre, R. (2008). Experience-dependent neural substrates involved in vocal pitch regulation during singing. *NeuroImage 40(4),* 1871–1887.

Zheng, Z., Munhall, K., & Johnsrude. I. (2010). Functional overlap between regions involved in speech perception and in monitoring one’s own voice during speech production. *Journal of Cognitive Neuroscience 22(8),* 1770–1781.

Appendix A

Western Research Ethics Approval



Western Research

Research Ethics

Western University Health Science Research Ethics Board
HSREB Delegated Initial Approval Notice

Principal Investigator: Dr. Scott Adams
Department & Institution: Health Sciences\Communication Sciences & Disorders,Western University

Review Type: Delegated
HSREB File Number: 109016
Study Title: Effects of Altered Intensity Feedback on Speech in Parkinson's disease.

HSREB Initial Approval Date: April 27, 2017
HSREB Expiry Date: April 27, 2018

Documents Approved and/or Received for Information:

Document Name	Comments	Version Date
Western University Protocol	Received April 24, 2017	
Recruitment Items	In-Class Recruitment Script	2017/04/24
Recruitment Items	CCAA Recruitment Script	2017/04/24
Recruitment Items	Telephone Script	2017/03/31
Letter of Information & Consent	Healthy Participants	2017/04/24
Letter of Information & Consent	PD Participants	2017/04/24
Data Collection Form/Case Report Form		2017/03/31
Other	Debriefing Script	2017/04/24

The Western University Health Science Research Ethics Board (HSREB) has reviewed and approved the above named study, as of the HSREB Initial Approval Date noted above.

HSREB approval for this study remains valid until the HSREB Expiry Date noted above, conditional to timely submission and acceptance of HSREB Continuing Ethics Review.

The Western University HSREB operates in compliance with the Tri-Council Policy Statement Ethical Conduct for Research Involving Humans (TCPS2), the International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use Guideline for Good Clinical Practice Practices (ICH E6 R1), the Ontario Personal Health Information Protection Act (PHIPA, 2004), Part 4 of the Natural Health Product Regulations, Health Canada Medical Device Regulations and Part C, Division 5, of the Food and Drug Regulations of Health Canada.

Members of the HSREB who are named as Investigators in research studies do not participate in discussions related to, nor vote on such studies when they are presented to the REB.

The HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.

[redacted]

Ethics Officer, on behalf of Dr. Joseph Gilbert, HSREB Chair
EO: Erika Basile ___ Grace Kelly ✓ Katelyn Harris___ Nicola Morphet ___ Karen Gopaul ___

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Curriculum Vitae
DONA ANITA ABEYESEKERA
School of Communication Sciences & Disorders, Health and Rehabilitation Sciences
Faculty of Health Sciences
University of Western Ontario

EDUCATIONAL BACKGROUND

Completed Degrees

2018	MClSc, Speech Language Pathology, Western University, London, Canada
2012	MSc. Psychology, University of Waterloo, Waterloo, Canada (Advisor: Katherine White)
2010	BEd, Urban Diversity (P/J Division), York University, Toronto, Canada
2008	BA, Psychology, York University, Toronto, Canada

Anticipated Degrees

2019	PhD, Speech and Language Sciences, Western University, London, Canada (planned completion August 2019) (Advisor: Dr. Scott Adams)

RELEVANT EXPERIENCE

Academic

Present	Assistant Professor, Buffalo State University, Buffalo, USA.
2018-present	Assistant Clinical Professor (Adjunct), McMaster University, Hamilton, Canada Foundational Knowledge and Clinical Skills Lab (Anatomy, Swallowing, Motor Speech, Voice Disorders)
2018	Lecturer, Western University, London, Canada Clinical Applications in Anatomy and Swallowing for Speech Language Pathology
2017-2018	Research Associate, Lawson Health Research Institute, London, Canada *Spasmodic Dysphonia Study*
2014	Research Assistant, Western University, London, Canada *Dr. Scott G. Adams, Parkinson Speech Treatment Study*
2012	Research Assistant, Speech and Stuttering Institute, Toronto, Canada *Dr. Arvind Namasiviyam, Pediatric Motor Speech Disorders*
2010-2012	Research Assistant, University of Waterloo, Waterloo, Canada *Dr. Elizabeth Nilsen, Communicative behaviour in children*
2010-2012	Research Assistant, Wilfrid Laurier University, Waterloo, Canada *Dr. Jeffery Jones, EEG study in Cognitive Neuroscience Lab*

Clinical

2018	Swallowing Clinical Practicum, Grand River Hospital, Waterloo, Canada
2017	Pediatric Practicum, Kidsability, Waterloo, Canada
2015	School Board Clinical Practicum, Waterloo District School Board, Waterloo, Canada
2013-2014	Neurological Disorders Clinical Practicum, H.A. Leeper Speech and Hearing Clinic, London, Canada
2008-2010	Clinician Assistant, Speech and Stuttering Institute, Toronto, Canada
2011-2012	Internship (and program development), Blue Balloon Health Services, Waterloo, Canada
2008-2009	Clinician Assistant and Program Support, Aphasia Institute, Toronto, Canada

HONOURS

2018	UWO Thesis Completion Fund (Academic merit) ($500)
2018	University of Western Ontario, CSD Teaching Assistant Award ($200)
2018	Faculty of Health Sciences Travel Award ($500)
2018	Health and Rehabilitation Sciences Travel Award ($250)
2017	University of Western Ontario, Graduate Studies Teaching Assistant Award Nomination
2016	Faculty of Health Sciences Travel Award ($400)
2016	Health and Rehabilitation Sciences Travel Award ($300)

TEACHING EXPERIENCE

Graduate

Winter 2018	McMaster Foundational Knowledge (SLP723), Assistant Clinical Professor
Fall 2018	McMaster Foundational Knowledge (SLP744), Assistant Clinical Professor
Fall 2018	McMaster Clinical Skills Lab (SLP742), Instructor
Fall 2018	UWO Clinical Applications in Anatomy and Physiology (9620Q), Instructor
2017	UWO Swallowing (CSD 9633), Teaching Assistant
2016-2017	UWO Anatomy and Physiology for Speech (CSD 9620), Teaching Assistant
Oct 11, 2017	UWO Anatomy (CSD 9620), Bones of the Skull Cadaver Lab (lead)
2016-2017	UWO Anatomy (CSD 9620), Independently organized extra support weekly review
2016	UWO Clinical Phonetics (CSD 9611), Teaching Assistant
2015-2016	UWO Neurologically Based Speech Disorders (CSD 9630), Teaching Assistant
Dec 7, 2015	UWO Neurologically Based Speech Disorders (CSD 9630), Invited Lecture

Undergraduate

Spring 2012	University of Waterloo Introduction to Psychology (PSYC 101), Teaching Assistant

Winter 2012	University of Waterloo Introduction to Psychology (PSYC 101), Teaching Assistant
2011	University of Waterloo Language Development (PSYC 320), Teaching Assistant

SUPERVISORSHIPS

Undergraduate research training and supervision - completed

2014	Tyler Stratton, Deep brain stimulation and its effects on Parkinson disease speech.
2016	Angeline Hong, Efficacy and acceptance of a low-cost Lombard-response device for the treatment of hypophonia in Parkinson’s disease.
2016	Jenny Zhang, Efficacy and acceptance of a low-cost Lombard-response device for the treatment of hypophonia in Parkinson’s disease.
2016	Eisha Gupta, Efficacy and acceptance of a low-cost Lombard-response device for the treatment of hypophonia in Parkinson’s disease.

LIFETIME RESEARCH FUNDING

Funded

2017-2019	*Peer Reviewed.* The role of auditory feedback for speech intensity regulation in Parkinson’s disease. A. Abeyesekera, S.Adams. Parkinson Canada Graduate Student Award, $40,000, National award.
2016	DBS Parameter Optimization for Voice Quality, Speech Intensity, and Prosody of Speech in PD. A. Abeyesekera, S. Adams. London Barbershop Harmony Society Award, $750.

Unfunded

2013-2017	Deep brain stimulation of the subthalamic nucleus on gait, tremor, and speech. M. Jog (PI), S., Adams (Co-PI), G. Gilmore, A. Abeyesekera, M. Delrobaei, C.Mancinelli, T, Knowles.
2016	Efficacy and acceptance of a low-cost Lombard-response device for the treatment of hypophonia in Parkinson’s disease. S. Adams (PI), M. Jog (Co-PI), N. Kumar, P. Rizek, A. Hong, J. Zhang, A. Abeyesekera, C. Mancinelli, T. Knowles.
2017	Voice quality severity and responsiveness to levodopa in Parkinson’s disease. D. Cushnie Sparrow, S. Adams, A. Abeyesekera, M. Pieterman, G. Gilmore, G, M. Jog.
2017-2018	Speech Effects of Spinal Cord Stimulation in Parkinson’s Disease. M. Jog (PI) A. Abeyesekera, S. Adams, O. Samoutos.
2017-2018	Speech task correlation between subthalamic nucleus and motor cortex in Parkinson’s Disease. M. Jog (PI), A. Abeyesekera, S. Adams, G. Gilmore

LIFETIME PUBLICATIONS

Peer Reviewed

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Gilmore, G., Delrobaei, M., & Jog, M. (2019). Deep Brain Stimulation Parameter Optimization for Voice Quality, Speech Intensity, and Prosody of Speech in Parkinson’s Disease. *Canadian Journal of Neurological Sciences. DOI: 10.1017/cjn.2019.16*

Cushnie-Sparrow, D., Adams, S., **Abeyesekera, A,** Pieterman, M., Gilmore, G., Jog, M. (2018). Voice quality severity and responsiveness to levodopa in Parkinson's disease. *Journal of Communication Disorders.*

Im, Hannah, Adams, S., **Abeyesekera, A.**, Pieterman, M., Gilmore, G., Jog, M. (2018). Effect of Levodopa on Speech Dysfluency in Parkinson's Disease. *Movement Disorders Clinical Practice.*

Knowles, T., Adams, S., **Abeyesekera, A.**, Mancinelli, C., Gilmore, G., & Jog, M. (2018). Deep brain stimulation of the subthalamic nucleus parameter optimization for vowel acoustics and speech intelligibility in Parkinson's disease. *Journal of Speech, Language and Hearing Research, 61(3).*

Abeyesekera, A. & White, K. (2012). The role of distributional information for learning phonemic contrasts in 14-month-old infants. MSc, CA.

Submitted and in-progress

Abeyesekera, A., Adams, S., Page, A., Jog, M. Effects of Altered Intensity Feedback on Speech in Normal Populations. *Canadian Acoustics*. *Revisions in progress 2019*.

Abeyesekera, A., Adams, S., Page, A., Jog, M. Review: Speech Intensity Regulation and Hypophonia in Parkinson's Disease. *Prepared for submission 2019*.

Adams, S., Kumar, N., Rizek, P., Hong, A., Zhang, J., **Abeyesekera, A.**, Mancinelli, C., Knowles, T., Jog, M. Efficacy and acceptance of a low-cost Lombard-response device for the treatment of hypophonia in Parkinson's disease. *American Journal of Speech-Language Pathology*. *Revisions in progress 2018*.

Abeyesekera, A., Adams, S., Gilmore, G., M., Jog, M. Speech task correlation between subthalamic nucleus and motor cortex in Parkinson's Disease. In progress.

Abeyesekera, A., Adams, S., Gilmore, G., M., Jog, M. Subthalamic nucleus neuronal encoding of speech in Parkinson's disease. In progress.

Abeyesekera, A., Adams, S., Samoutos, O., Jog, M. Speech Effects of Spinal Cord Stimulation in Parkinson's Disease. In progress.

Abeyesekera, A., Adams, S., Cushnie-Sparrow, D., Pieterman, M., Jog, M. Pharmacological treatment effects on Parkinsonian speech intensity. In progress.

Abeyesekera, A., Adams, S., Page, A., Jog, M. The role of auditory feedback for speech intensity regulation in Parkinson's disease. In progress.

PRESENTATIONS AT MEETINGS

Invited

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Gillmore, G., Delrobaei, M., Jog, M. Effect of different deep brain stimulation parameters on voice quality, speech intensity, and prosody in Parkinson's disease. Oral presentation at **Journal Club Conference, Wilfrid Laurier University, Canada, 2017.**

Contributed (Peer reviewed)

Abeyesekera, A., Adams, S., Page, A., Jog, M. The role of auditory feedback for speech intensity regulation in Parkinson's disease. Poster presented at **Motor Speech Conference, Georgia, United States of America, 2018.**

Cushnie-Sparrow, D., Adams, S., Abeyesekera, A., Pieterman, M., Gilmore, G., Jog, M. Effects of levodopa on voice quality in Parkinson's disease. Poster presented at **Motor Speech Conference, Georgia, United States of America, 2018.**

Abeyesekera, A., Adams, S., Samoutos, O., & Jog, M. *Speech effects of spinal cord stimulation in Parkinson's disease.* Oral presention at **Movement Disorders Research Retreat, London, Canada, 2017.**

Mancinelli, C., Adams, S., Abeyesekera, A., Knowles, T., Delrobaei, M., & Jog, M. *Effects of Deep Brain Stimulation Parameter Settings on Rate of Speech in Parkinson's Disease.* Poster presented at the **American Speech–Language–Hearing Association, Pennsylvania, United States of America, 2016.**

Knowles, T., Adams, S., Abeyesekera, A., Mancinelli, C., & Jog, M. *Deep Brain Stimulation Parameter Optimization for Speech Intelligibility and Vowel Acoustics in Parkinson's Disease.* Presented at **International Clinical Phonetics and Linguistics Association Conference, Nova Scotia, Canada, 2016.**

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Delrobaei, M., & Jog, M. *Deep brain stimulation and speech in Parkinson's disease: Effects of stimulator settings and electrode contact positions.* Poster presented at **Southern Ontario Neuroscience Association, Waterloo, Canada, 2016.**

Knowles, T., Adams, S., Abeyesekera, A., Mancinelli, C., Delrobaei, M., & Jog, M. *Deep Brain Stimulation Parameter Optimization for Speech Intelligibility and Acoustics in Parkinson's Disease.* Poster presented at **Motor Speech Conference, California, United States of America, 2016.**

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Delrobaei, M., & Jog, M. *Deep Brain Stimulation Parameter Optimization for Voice Quality, Speech Intensity, and Prosody of Speech in Parkinson's Disease*. Poster presented at **Motor Speech Conference, California, United States of America, 2016.**

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Delrobaei, M., & Jog, M. *Effect of Different Deep Brain Stimulation and Speech.* Oral presention at **Movement Disorders Research Retreat, Toronto, Canada, 2016.**

Abeyesekera, A., Adams, S., & Jog, M. *Instantaneous auditory feedback of speech intensity and hypophonia in Parkinson's disease: A pilot study among healthy controls*. Oral presentation at **Health and Rehabilitation Science Graduate Research Forum, London, Canada, 2016.**

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Delrobaei, M., & Jog, M. *Effect of Different Deep Brain Stimulation Parameters on Voice Quality, Speech Intensity and Prosody in Parkinson's Disease.* Oral presention at **Movement Disorders Research Retreat, London, Canada, 2015.**

Abeyesekera, A., Adams, S., Mancinelli, C., Knowles, T., Delrobaei, M., & Jog, M. *Effect of Different Deep Brain Stimulation Parameters on Voice Quality, Speech Intensity and Prosody in Parkinson's Disease.* Poster presented at **Health and Rehabilitation Graduate Research Forum, London, Canada, 2015.**

Knowles, T., Adams, S., Abeyesekera, A., Mancinelli, C., Delrobaei, M., & Jog, M. *Effect of deep brain stimulation parameters on speech intelligibility and speech acoustics in Parkinson's Disease.* Poster presented at the **Health and Rehabilitation Graduate Research Forum, London, Canada, 2014.**

Mancinelli, C., Adams, S., Abeyesekera, A., Knowles, T., Delrobaei, M., & Jog, M. *Deep brain stimulation parameter optimization for rate of speech in Parkinson's Disease.* Poster presented at the **Health and Rehabilitation Graduate Research Forum, London, Canada, 2014.**

Abeyesekera, A., Adams, S., Mancinelli, C., Rahimi, F., Delrobaei, M., & Jog, M. *Deep Brain Stimulation Parameter Optimization for Speech in Parkinson's Disease*. Poster presented at **Southern Ontario Neuroscience Association, London, Canada, 2014.**

Abeyesekera, A., Adams, S., Mancinelli, C., Rahimi, F., Delrobaei, M., & Jog, M. *Deep Brain Stimulation Parameter Optimization for Speech in Parkinson's Disease*. Poster presented at **ARGC/Faculty of Health Science Symposium, London, Canada, 2014.**

Abeyesekera, A., Adams, S., Mancinelli, C., Rahimi, F., Delrobaei, M., & Jog, M. *Optimal Deep Brain Stimulation Settings for Speech in Parkinson's Disease*, Poster presented at **HRS graduate Research Forum, London, Canada, 2013.**